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VOLUME X.

NERVOUS AND MENTAL DISEASES

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NERVOUS DISEASES.

SYMPTOMATOLOGY.

The Co-ordination of Single Muscular Movements in the Central Nervous System. C. E. Beevor[1] in his address before the Neurological Section of the American Medical Association at Chicago in June, 1908, discussed the coordination of a single muscular movement, by which he means the single movement of one joint, such as flexion of the thumb, or of several similar joints, such as flexion of all the fingers and the thumb, as in grasping. In the action of grasping (1) we have first the muscles which do the work, viz., the flexors of the fingers and thumb, which are called the prime or principal movers. (2) We also have the antagonists to the movement, viz., those muscles which produce a movement opposite to the one we wish to perform, which in this case are the extensors of the fingers and thumb. (3) There are, next, the synergic muscles, so called because they work with the prime movers whenever these muscles pass over two joints, such muscles neutralizing the action of the muscles not required. For example, the flexors of the fingers in their course from origin to insertion pass the wrist-joint; and it is evident that after these muscles have flexed the thumb and fingers they will also tend to flex the wrist. But such flexion of the wrist would be awkward, and is prevented by the contraction of synergic muscles which are the extensors of the wrist. These may be felt to contract when the fingers are flexed. (4) Lastly, there are the fixation muscles, which fix the joint and prevent movement between the joint in which the required movement is being performed, and the trunk. Thus, in flexing the fingers against resistance, the tendency of the resistance is to extend the wrist and elbow; and this is counteracted by the flexors of the wrist and the flexors of the elbow—the biceps, supinator longus, and other muscles. Thus there are four sets of muscles taking part in a single muscular act: (1) prime or principal movers; (2) synergic muscles; (3) fixation muscles; (4) antagonists.

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(1) Jour. Am. Med. Assoc., July 11, 1908.

Prime or Principal Muscles of Motion. Observation indicates that the prime muscles do not all contract together, but, instead, that they contract in a sequence which is always preserved. Moreover, no muscle in the body known to Beevor ever takes part as a prime mover in two actions diametrically opposed to each other, unless possibly it be the latissimus dorsi alone. Duchenne, from electrical stimulation, concluded that the clavicular fibers of the pectoralis major are both elevators and depressors of the humerus, and this Beevor disputes, saying that they are never depressors.

Synergic Muscles. In extending the thumb we simultaneously contract the ulnar carpal muscles, and this contraction may easily be felt while the thumb is being extended. These ulnar carpal muscles are therefore synergic muscles. Again, in strong flexion of the hip when the knee is bent, the knee would tend to be extended by the rectus femoris and rotated in by the sartorius, if it were not for the synergic contraction of the short head of the biceps, which prevents such undesired movements. "The relation between the prime movers and the synergic muscles is very close, and it seems impossible to contract one without the other. For instance, it is not possible to extend the thumb in the slightest degree without causing a contraction of the extensor and flexor carpi ulnaris, and if the prime mover be paralyzed by a lesion of the peripheral nerves the synergic muscles go on contracting and produce movements which are not intended."

Fixation Muscles. "If the wrist be in the position of pronation, and the fingers be extended against resistance, this resistance, if strong enough, will tend to overcome the wrist and cause it to flex. So, to prevent the wrist from being flexed, the extensors of the wrist take part in the movement, and to prevent the elbow being extended by the pressure the biceps contracts. It is to be observed that the extensors of the wrist and the biceps as fixation muscles do not come into action at once, but that a certain amount of resistance has first to be experienced; it seems, however, that as soon as the joints begin to be displaced by the extra pressure, then the fixation muscles contract.

"Another instance of fixation muscles is the contraction of the muscles of the upper arm, which occurs when the fingers and thumb flex in the movement of grasping an object. The flexors of the fingers, arising as they do from the internal condyle of the humerus, have a slight action of flexing the elbow; and, especially when the elbow is at a right angle, to prevent this flexion and to steady the elbow-joint; the triceps contracts; but, like other fixation muscles, it does not contract until a certain amount of displacement takes place. In my own case I can not feel the triceps contract until the dynamometer has registered a grasp of from six to ten pounds (3-5 kgs.)."

The Antagonists. Beevor believes that in any given movement the antagonists relax, and he has used such relaxation on several occasions in differentiating hysteria from organic disease of the nervous system. In hysteria there seems to be a definite inability to inhibit the antagonists. "About five years ago I described the following condition which occurred in a case of functional paralysis in a girl: She had partial hemiplegia, and when she was told to extend the affected elbow against resistance, in place of the extensor muscles contracting, and the antagonists—biceps, brachialis anticus and supinator longus—relaxing, the antagonists actually contracted first, and the first movement took place in the biceps and supinator longus. This was followed by contraction of the triceps and again by the biceps, giving rise to the to-and-fro hesitating movement which is so characteristic of attempted movements in functional cases. Since seeing this first case I have come across many cases of functional paralysis showing the same inability to inhibit the antagonists."

As to the question of the part of the nervous system in which these groups of muscles are coördinated to form a movement, as between the spinal cord and the motor cortex, Beevor inclines to the former. He likens the spinal cord to an engine room on an ocean liner. "The captain on the bridge sends down messages to the engine room and his orders are put into action by the engineer, but the captain has nothing to do with the mechanism of turning on the particular steam cocks, etc. The captain also gives messages to the man at the wheel, who puts in action the steam steering gear. The captain on the bridge represents the cerebral mechanism, including the motor area, and the engineer represents the posterior cornual cells and turns on the proper amount of steam. The captain does not know what is going on in the engine room, except by his orders being properly carried out, and likewise the brain knows nothing about how the movements are done. If the ship have twin-screws and be driven by turbines, then the engines for going astern will constitute the antagonists to the prime movers for going ahead. The rudder will represent the synergic muscles, and if it be required to turn the ship to the right the rudder will be used to counteract the action of the right screw, which is not required. If the rudder does not act, the ship performs a movement which the captain did not intend, and if the synergic muscle be paralyzed, the movement, which the brain ordered, is wrongly carried out, and neither the captain nor the brain knows that it will happen, as the block takes place either in the engine room or at the periphery from the breaking of a screw shaft, i. e., in the anterior horns or in peripheral nerves.

"On the other hand, if the mechanism for coördinating the muscles took place entirely in the brain, it would mean that the captain would have to manage all the machinery of the engine room—which would have to be on deck—and would himself have to turn on the necessary steam cocks, and at the same time he would have to steer the ship himself."

Mendel's Dorsal Foot Reflex. The reflex known by this name was first described by Kurt Mendel in 1904,[1] and is obtained by tapping the dorsum of the foot. There follows dorsal flexion of the toes from the second to the fifth, inclusive, in healthly individuals, but plantar flexion

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(1) Neurol. Centralbl., No. 5.

in organic disease of the nervous system. The reflex is thus comparable to the Babinski toe-sign, and has the same diagnostic value. As a rule the dorsal foot reflex in plantar flexion goes hand in hand with Babinski's toe-sign; but it has been observed by Mendel in 18 cases in which the Babinski toe-sign was *not* present. Thus in these cases it proved a more delicate and early sign than Babinski's toe-sign.

To these 18 cases O. B. Meyer[1] adds 10 more, all showing Mendel's plantar flexion response without dorsal flexion of the great toe. In one case, encephalitis pontis, hysteria might well have been suspected had not Mendel's sign been present. The total of 28 cases were distributed as follows: 6 of myelitis, 4 of multiple sclerosis, 8 of hemiplegia, 4 of disease of the pons, 2 of infantile cerebral paralysis, 1 of tumor of the spinal cord, 2 of Brown-Séquard paralysis, and 2 of progressive paralysis. Interesting is a case of multiple sclerosis in which Babinski's toe-sign, Oppenheim's sign, and Mendel's reflex were all present at some time during the course of the malady, but only one sign at a time, never two or three at once.

The Mendel reflex, or, as the author calls it, the Mendel-Bechterew dorsal foot-reflex, has also been thoroughly studied by E. Osann.[2] His conclusions follow:

1. The normal extension reflex of Mendel is a so-called idiomuscular reflex and is caused by direct irritation of the extensor brevis digitorum. This reflex is almost invariably present in normal individuals. It may be inhibited in whole or in part by external influences, such as edema, changes in the joints, voluntary tension of the joint extensors, and atrophy of the muscles.

2. Absence of the extension reflex alone is of no special diagnostic significance; still it has a certain value when not due to any of the above-mentioned external influences, in that it ushers in the pathologic flexor reflex in the more pronounced cases. The absence or presence of the extension reflex therefore serves to a certain extent as a measure of the intensity of the Mendel-Bechterew reflex, so that

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(1) Berl. klin. Woch., Aug. 26, 1907.
(2) Muench. med. Woch., Dec. 10, 1907.

one may distinguish two degrees of the latter, a completely positive and a partial Mendel-Bechterew reflex.

3. The Mendel-Bechterew reflex occurs in cases of organic spastic paresis of the lower extremities. It is present in many cases in which Babinski's toe-sign is present; still it is not so comprehensive a reflex as the latter. In a few cases a positive Mendel-Bechterew reflex occurs when Babinski's toe-sign is absent.

4. Despite the fact that both reflexes are commonly simultaneously present, there is no essential similarity between the two. While Babinski's toe-sign is a pure skin reflex independent of the tone of the muscles of the lower extremities, the Mendel-Bechterew dorsal foot reflex depends for its existence upon the presence (often slight) of a hypertonic condition of the muscles of the foot and toes. In a strikingly large number of cases the Mendel-Bechterew sign is coexistent with ankle-clonus.

5. There is a perfectly clear analogy between the Mendel-Bechterew reflex and the flexor reflex of the fingers described by v. Bechterew.

Amnesia. The various amnesias are discussed by M. R. Benon.[1] Memory is a general property of the nervous centers, by virtue of which they preserve and reproduce the sensations which have produced them. Memory is multiple; there is no one memory, but there are memories. They are the foundation of consciousness and of individuality. Without memory we could hardly be individual units. The following table of the clinical characteristics of amnesias is presented by the author:

A. Amnesia with reference to its causes and the time it involves.
    (1) Retrograde: amnesia, for example, in which memory of everything before the accident causing the amnesia, is wiped out.
    (2) Anterograde: amnesia dating from the accident causing it, involving everything happening subsequent to the accident. According to Janet, this amnesia is one of assimilation, or defective mental synthesis.

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(1) Gaz. des Hôp., June 13, 1908.

    - (3) Retro-anterograde: amnesia comprising all memories before and after the exciting cause of the amnesia.
- B. Localization of amnesia with respect to an individual's life.
    - (1) Amnesia which is localized, being limited to a particular period of time.
    - (2) Amnesia which is not definitely limited between certain dates, but is only vaguely delimited.
- C. Quantitative changes of memory.
- D. Qualitative defects of memory.
    - (1) General.
    - (2) Special.
        - (a) Visual amnesia; psychic blindness, word-blindness.
        - (b) Auditory amnesia; word deafness. Musical amnesia.
        - (c) Tactile amnesia; astereognosis.
        - (d) Olfactory amnesia; anosmia.
        - (e) Gustatory amnesia.
        - (f) Generalized sensory amnesia; generalized agnosia.
        - (g) Anesthetic amnesia.
        - (h) Motor amnesia; apraxia.

It is not necessary to follow the author's amplification of this outline, which is very complete, though slightly condensed, in the above table. Among the general qualitative defects of memory should be the paramnesias, or recollections of false memories or imaginations, or things which never happened. They ought rather to be called pseudamnesias or false remembrances, and should be classed among illusions and perversions of memory rather than in the amnesias or defects of memory.

A clinical examination as to amnesia is made somewhat as follows: (1) Ask the patient his full name, occupation, and age. (2) Next question him as to events of the present year, month, week, date, hour, or season, and then as to events of the past, his year of marriage, the memories of that year, etc. (3) Next question him as to places with which he is at present familiar, his home, shop, neighborhood; and then as to places with which he was long ago associated, such as the place where he was born, or married, or where he lived in early life. (4) Having settled the question of the memory in both time and place, next inquire into actual facts, recent or remote, individual or relating to the family, professional or social, immediate relatives and antecedents. These questions will uncover any memory defects present. If the patient has been a student he should be questioned as to his educational attainments, and in any event it is well to test the memory by mental arithmetic, spelling and writing, geography and history. To be sure, these are mental tests in a way, but they aid in uncovering amnesias. An excellent bibliography accompanies the article.

Apraxia. The subject of apraxia is very thoroughly discussed by Félix Rose[1] in a lengthy paper. The word means an inability to execute an act directed toward a certain end, but not by reason of paralysis or ataxia. An act apparently voluntary is in the last analysis reflex, and the arc has three portions: the psychosensorial pathway, the intrapsychic pathway, and the psychomotor pathway. Apraxia proper results only from interruption of the psychomotor pathway. Interruption of the intrapsychic pathway constitutes mental alienation. There is, however, so continuous an interplay between the psychosensory and the psychomotor phenomena, that pure motor apraxia hardly exists in practice. Practically, therefore, apraxia consists in a disturbance of voluntary movements, manifesting itself by the impossibility of executing an act directed toward a given end, this disturbance not being ascribed either to paralysis, ataxia, agnosia, or mental defect.

The first case of apraxia was reported by Liepmann in 1900 in a man who used his right hand in an absurd manner, and so passed for a dement. When obliged to use his left hand, however, he used it intelligently. Investigation showed his intelligence to be sufficiently well conserved, and there was no word- or mind-blindness or visual disturbances. The only thing involved seemed to be the deep sensibilities of the right hand. The defect was attributed by Liepmann to the center for motor representa-

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(1) La Semaine Méd., April 22, 1908.

tions, which he placed in the third frontal gyrus, the white matter of the supramarginal gyrus, and the superior and inferior parietal gyri. These latter localizations were made because of apparent involvement of the visual and auditory memory centers. The postmortem examination proved Liepmann to be correct.

After this case numerous others have been reported, Liepmann himself furnishing a second; and Rose reviews at length the cases of Herzog, Abraham, Marcuse, Strohmayer, Westphal, Hollander, Bonhoeffer, Kleist and Pick. Analysis of these cases shows that under the name of apraxia three different types have been described, all meeting on the common point of being psychomotor phenomena. These three types are the motor (Kleist), the ideomotor (Liepmann), and the ideational (Pick). According to Liepmann the psychic mechanism of apraxia works out as follows: An idea of an end to be attained or an act to be accomplished, is composed of a series of ideas relating to the different successive movements necessary to that act, and these may be called the ideas of the partial end. The sum total of all of these ideas makes up the kinetic formula for the act. The disturbance of this kinetic formula may be due to pure motor apraxia, to ideational, or to ideomotor apraxia. In the ideational type there is an omission of a more or less large number of these "ideas of the partial end," or there is an inversion or interversion in their regular sequence. The act appropriate to one of these misplaced "ideas of the partial end" is then executed perfectly, but, being ill-timed, is really apraxic. The kernel of the matter lies in a disturbance in the association of ideas.

In ideomotor apraxia the terminal act is not adequate to the end proposed for other reasons. In each "idea of the partial end" there are several factors, the optic representation of the movement, its abstract kinesthetic representation, and its specific kinesthetic representation by a given member. In ideomotor apraxia there is a gap between the optic and abstract kinesthetic representations, on the one hand, and the specific kinesthetic representation, on the other. Thus the act executed is not logical either in general or in relation to its object. In pure motor apraxia the muscles do not obey the will, the motor pathways have been disturbed; in ideomotor apraxia they obey the will, but the will fails to regulate the proper succession of the elementary acts. This is preëminently true in ideational apraxia; so that this form is nearer agnosia than the other two forms. In ideomotor apraxia, substitution of movements proper for one purpose, for a purpose for which they are not fitted, occurs. In ideational apraxia the movements executed are proper for no purpose whatever. The whole matter of apraxia is further complicated by the tendency of the patient to fall back constantly upon the movement last executed. This has been called intoxication by an idea, and may be clonic, a repetition of the same movement, or tonic, a tendency to prolong the muscular innervation. In practice it is the ideomotor apraxia which is most met with, and it is just as difficult to study as is aphasia.

Can the site of the lesion causing apraxia be localized? The postmortem examinations are contradictory, lesions having been found in the following gyri: third frontal, supramarginal, superior parietal, corpus callosum, in two cases; and in the inferior parietal, middle third of the ascending parietal in a third. In a case of Monakow's there was a tumor of the lenticular nucleus compressing the whole hemisphere, and in a case of Westphal's an internal hydrocephalus. Here is found, for example, a softening of the middle third of the corpus callosum, and there gummata of the caudate nucleus associated with superficial gummata. Thus there is no unanimity. Monakow believes that apraxia demands for its existence the involvement of very extensive cerebral territories, just as does aphasia, and he shows that softening of the inferior parietal and of the first temporal convolutions did not prevent recovery from apraxia following apoplexy. Very curious is the fact that a patient with right hemiplegia may show a pronounced left-sided apraxia. This is supposed to show the relationship of the corpus callosum to apraxia, but Monakow has reported a case of hydrocephalus with marked atrophy of the corpus callosum, presenting no apraxia.

Rose reports in this paper the case of a man having diffuse meningo-encephalitis, with Jacksonian convulsions limited to the face and the left arm, an apraxia of the left hand, and a bilateral apraxia of the muscles innervated by the cervico-facial division of the seventh nerve, and of the muscles of mastication and of the tongue. Everything cleared up within five days. His apraxia could not be ascribed to an intellectual deficit, because movements ordered were properly executed with the right hand, though improperly with the left. There was no mind-blindness or deafness, though at times there was some slowness in recognizing objects seen. There was no ataxia. The apraxia seemed ideomotor, due to involvement of the motor pathway, yet there was no paresis. The case apparently shows that a lesion of the right hemisphere may produce a bilateral apraxia of the muscles of the face, tongue, and inferior maxilla, at least in certain of their movements. Although for unilateral movements of the limbs and certain bimanual manipulations, the right hemisphere may exert no influence over the limbs of the right side, nevertheless, for certain motor functions habitually bilateral, particularly for the muscles of mastication and those of the tongue and face, surely the integrity of both hemispheres and their association pathways is necessary.

Aphasia. F. W. Mott[1] publishes the report of a case of bilateral lesion of the auditory cortical center, with complete deafness and aphasia. The patient was stone-deaf, and yet understood simple written sentences. For example, she was shown the written question, "How many children have you had?" and the numerals 0, 1, 2, 3, 4 were written down, at which she showed signs of dissatisfaction. Mott then wrote down "miscarriages 2," at which she became very excited and said "is that." She was only partially word-blind, after fatigue. Spontaneous writing was almost impossible. She undoubtedly reasoned, looked to be intelligent, and was clean in her habits. She recognized Mott after the interval of a year had elapsed since his last visit to her. After her death there were found bilateral areas of atrophy of both auditory centers. Mott therefore disagrees with Marie as to aphasia being due to lessened intelligence. "If we consider her acts, her endeavors to make people understand her own wants, and her desire to help

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(1) Brit. Med. Jour., Aug. 10, 1907.

the attendants when the other patients were getting into mischief, also the fact that her habits were clean, that she recognized me after a year's interval—everything shows that she reasoned, and that the mental impairment, although undoubtedly great, was more apparent than real. She comprehended her surroundings, and reasoned and acted to the best of her ability in response to the stimuli arriving in her cerebral cortex by the sensory avenues of intelligence still open. Considering the enormous amount of cerebral destruction as shown by the loss of brain weight, her mental condition compared most favorably with the average asylum patients whose brains seldom lose anything approaching the amount of loss by this patient, which must have been quite one-fourth of the total brain weight.

"The case I have reported, in my opinion, supports Bastian's views that the primary revival of spoken words during thought takes place in the great majority of persons by a subconscious process in the auditory center, for word-deafness can alone explain the loss of speech in the first attack; and instantly that the patient became absolutely deaf from the second lesion, she again became dumb and remained so. The subconscious revival of word images precedes the correlated revival of the complementary kinesthetic images, and these again successively incite the associated groups of neurones in the bulbar centers. It has been shown by many observers, commencing with Stricker, Galton, Ribot, and others, that different persons think in different ways, according to the predominance of the visualizing, auditory, kinesthetic (or motor) faculties; a large proportion of individuals are, however, indifferents.

"The fact that she was unable spontaneously to initiate written speech, although her ability to read and to understand simple sentences (when I saw her) and to name correctly objects by means of printed or written letters and words, showed that her visual word-center was not entirely destroyed. This suggests to my mind the correctness of the hypothesis that the *primary* revival of the articulation of words in her case took place in the auditory centers, and I would agree with Bastian that in *motors* *visuals,* and *indifferents* the primary revival of words takes place in that portion of the brain in which (in a child) speech first develops. In the child, words are first learned by hearing certain sounds associated with certain objects, and simple thoughts connected therewith are acquired before the child has the power of articulating them. He sees an object before he can name it; he must revive those auditory impressions which he had previously heard associated with it, otherwise how can we explain the fact that a child in full possession of speech even as late as the fifth, sixth or seventh year, if he becomes deaf will certainly become dumb, unless he is trained by lip-reading—that is, unless the primary incitation to articulate speech be transferred from the auditory to the visual word-centers? The facts related in this case do not accord with the statement of Bernard that the motor center of speech may become independent of the sensorial center which had presided over its education."

E. N. von Mayendorf,[1] in a paper on the localization of the motor speech center, after recounting the work of Marie and others upon the subject, concludes from his own studies that the third inferior frontal gyrus is anatomically and physiologically merely the lowest part of the anterior central gyrus, and that Pierre Marie is right in denying that it has any part in the causation of motor aphasia.

Location of Motor Cortex. Rossi and Roussy[2] report three cases of amyotrophic lateral sclerosis with degeneration of the pyramidal tracts, which cases throw distinct light upon the question, now practically settled, of the location of the motor cortex. The work of Sherrington and Grünbaum, at first, and that of many other observers subsequently, shows the motor cortex to lie wholly in front of the fissure of Rolando. The three cases here reported were carefully studied, and the cords were stained both by the Marchi and the Weigert methods. In the ascending frontal convolution was found a degeneration of the radiating fibers of the corona radiata, clearly evident by the Marchi method, and even more plainly by the Pal method.

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(1) Berl. klin. Woch., Aug. 10, 1908.
(2) La Semaine Méd., Aug. 14, 1907.

The cells of Betz were distinctly altered. In the ascending parietal gyrus, on the other hand, both cells and fibers were normal.

NEUROSES.

HYSTERIA.

Nature of Hysteria. The nature of hysteria has been engaging the attention of the Paris Neurological Society during the past year, and a summary by H. Meige[1] of the opinions of the various members may be of value. Déjérine, Crocq, Pitres and others declare that there are other affections than hysteria, such as neurasthenia, psychasthenia, and obsessions, in which one may observe accidents caused by suggestion, or cured by it, exactly as in hysteria. Babinski, Brissaud, Ballet, Dupré, Souques, and Meige declared that such cases do not exist. "Nervous pathology," says Déjérine, "abounds with examples of subjects, who, made ill by suggestion and cured by persuasion, are not hysterics." He cites cases of stomach trouble caused by the suggestion of one physician and cured by that of another. How shall these cases be distinguished from hysteria, then? In that they do not show the stigmata of hysteria or their peculiar mental state. The neurasthenic is constantly preoccupied with his condition; the hysteric troubles himself very little about it, and sometimes not at all.

Babinski, however, believes that these stigmata are of very limited diagnostic value, and he will not admit the peculiar mental state mentioned by Déjérine as a distinctive symptom, calling attention to the fact that many hysterics harp upon their ailments day and night. Dupré also attacked Déjérine's view, affirming that there were many hypochondriacs, many possessed with obsessions, many nosophobiacs, who were not and could not be helped by any amount of suggestion. Brissaud, Ballet, Souques, and Meige also assailed Déjérine's statement. In the discussion the weight of opinion favored the conclusion that, outside of hysteria, there are no morbid states at present well defined, in which one may meet disturbances presenting exactly the same characters as hysteria itself.

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(1) La Presse Méd., July 25, 1908.

Attempts to define hysteria led to various results. According to Babinski, "Hysteria is a psychic state rendering the subject who finds himself in it, capable of autosuggestion. It is manifested chiefly by primary disturbances, and accessorily by a few secondary disturbances. Characteristic of the primary disturbances is the fact that it is possible to reproduce them by suggestion with rigorous exactness in certain subjects, and to cause them to disappear under the influence of persuasion alone. Characteristic of the secondary disturbances is the fact that they are strictly subordinate to the primary disturbances." Ballet was of the opinion that hysteria is characterized less by the disturbances suggested than by the psychic change, emotional in essence, which makes such an aptitude for suggestion possible. If hysteria is to be defined, this mental state must be analyzed and defined. "I have defined hysteria," says Crocq, "as a psychopathologic state characterized by hyperimpressionability, diminution of cerebral control, and hypersuggestibility. I do not conceal from myself the fact that this definition is a little vague, but I prefer its imperfection to a premature scheme of hysteria."

At the French Congress of Alienists and Nerologists, held at Geneva and Lausanne in August, 1907, Claude, and also Schnyder,[1] read papers bearing upon the nature of hysteria. Claude finds it very hard to define the disease. The hysterical individual lacks the control of the ego over his perceptions and conceptions. As Raymond says, a series of correcting sensations of psychic, motor, sensory, tactile, muscular, articular, or others, not being at the disposal of the subject, because they are not perceived, there results the fact that certain perceptions, which are clearly registered, assume a strength and scope so exaggerated as to be pathologic. The consequence of this psychic perturbation is the development of suggestibility, which, carried to extremes, permits to burst forth in all their intensity the innumerable manifestations of the neurosis, all of these being really in harmony with the patient's thoughts, mental representations, and sensations, especially of emotional origin. Raymond has shown that this conception accounts

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(1) La Semaine Méd., Aug. 14, 1907; Gaz. des Hôp., Aug. 17, 1907.

for all the symptoms generally called hysterical, and he has therefore been led to summarize his conceptions of hysteria as follows: "The psychoneurosis hysteria, which is only a special manner of reacting and of feeling on the part of the individual involved, results from a particular modification of the nervous dynamism, characterized by a disturbance in the realm of the cortical or subcortical reflexes. These, whether inhibited or excited, call forth the dissociation of the physio-psychologic operations, and lead to their acting alone and in isolation; thus there are created nervous syndromes, more or less lasting, in the different domains which these reflexes control."

Clinical observation indeed shows that there is a group of individuals who from childhood present manifestations of what is ordinarily called *nervousness*. This condition, usually inherited, sometimes created by bad hygiene, some accident, or a severe illness, is characterized by faulty regulation of the reflex processes which intervene in the organic and psychic functions, or which bring the former into relation with the latter. As to the organic functions, this nervousness is revealed by reactions either excessive or not appropriate to the cause which has aroused them; they are, in general, characterized by exaggeration or suppression. As to the psychic functions, the nervousness is revealed by exaggeration of the emotivity or the sensitivo-sensorial receptivity, or by apathy and indifference. These attributes of nervousness are preserved, amplified and transformed by hysteria. Because of his organic nervousness the hysteric is endowed with a sensory hyperesthesia which is responsible for a multitude of sensations of varying intensity, and at the same time he is unable to correct or correlate them. Certain impressions or sensations thus assume an enormous importance, and on this substratum of nervousness, give rise to a whole train of exaggerated reactions. Thus, in earliest childhood, emotion, fear, pain, which ought to betray themselves by crying or facial expression, express themselves in convulsions, vasomotor disturbances, even syncope. In later life the intellectual functions (judgment, reason, generalization, etc.) are likewise disturbed, since they have for their foundation the aforesaid sensitivo-sensorial impressions. Ultimately these judgments, false conclusions and generalizations may occupy the consciousness so completely as to crowd all else out.

As to the genesis of the different symptoms or "accidents" of hysteria, Claude believes them to have as a common characteristic an excessive variability in the importance of the conscious or unconscious perceptions and apperceptions of all sorts, so that some assume a dynamogenic function, while others are annihilated, thus leading to the dissociation of psychic synthesis. Hysteria may then be considered as a diathesis originating in a constitutional defect of the nervous system, called nervousness, and is characterized by faulty regulation of the elementary psychic and organic reflex processes.

Schnyder, of Berne, believes that it is no longer possible to attribute to hysteria innumerable symptoms formerly so ascribed. Most of these symptoms are really those of various psychoneuroses. The psychic conditions favorable to the development of hysterical troubles are a fantastic imagination, a mental irrationalism leading the individual to an inexact notion of realities, to an abnormal expression of his affective states, and finally to a more or less complete change in his personality. Hysterical states really are derived from ordinary mentality by insensible gradations. The modification of the mentality called hysterical is characterized by exaggeration and the perversion of the physical and psychophysical reactions ordinarily met with in the normal. In particular, there are emotional reactions and manifestations of suggestibility. In a general way one may say that the hysterical modification of the mentality has for its point of departure a defect in judgment, leading to an erroneous conception of real situations, and to defective adaptation. Hysteria is, above all, a disease of evolution of the human mind; it represents a breach in the development of mentality. So hysteria in its pure form is a disease of young individuals, as it is also the disease of the childhood of the human race. This is why it was so prevalent in the middle ages, because it manifests itself in individuals whose mentality represents a primary stage in the mental evolution of the human race. For this reason hysteria is common in the rural population and in the working and peasant classes. And the migration of country people to the cities, by reason of the efforts towards adaptation which are forced upon them, constitutes an important cause of modern hysteria. Hysteria is the ordinary response to the exigencies of life among the uprooted or disenchanted who still present the simplicity of psychic reactions met with in the child.

Some regard hysteria as a manifestation of mental degeneracy. Not so Schnyder. He admits that such degeneration may give rise to symptoms resembling those of the hysteric, but they are essentially different. Such degenerative forms are met with in the upper classes, and are to be regarded as accidental variations from the normal.

In conclusion Schnyder makes the following summary: "Hysteria represents a morbid condition of the mentality, and belongs to the category of psychoneuroses. The hysterical modification of mentality depends upon a lack of judgment and of reasoning power, a peculiarity which underlies the child's mentality, but which in the adult may be the result of degeneration. The faulty judgment betrays itself in a fantastic appreciation of realities, in an erroneous moral conception of life. It leads to an abnormal expression of emotional states, to dissociations of the personality, to the production of subconscious acts, and to phenomena having the character of autosuggestions, which may be followed by abnormal psycho-physical reactions.

The nature and etiology of hysteria are ably discussed by L. Alquier.[1] According to Lasèqne, as called to mind by Babinski, hysteria is like a waste-paper basket into which are thrown all papers which cannot be readily classified. As a matter of fact, there is not a symptom of morbid pathology which hysteria cannot reproduce. At present the whole subject is being revised, and new researches, carefully guarding against unconscious suggestion, are being made. At the discussion before the Paris Neurological Society all present readily admitted that the stigmata of hysteria are really due in large part to suggestion. For example, the so-called hysterical ocular stigmata, dyschromatopsia can no longer be found in hysterical subjects, according to Brissaud, when the dip]oscope is used. The

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(1) Gaz. des. Hôp., Aug. 8, 1908.

same is true of polyopia or diplopia monocularis. Narrowing of the field of vision is not found when suggestion is avoided. Indeed, there are no ocular symptoms peculiar to hysteria.

Discussion concerning sensory disturbances, paralyses, and contractures led to the conclusion that these symptoms also are most often due to suggestion. In a recent study of clonus in which tracings were made, Claude and Rose have shown the organic clonus to be even and regular, while the hysterical clonus is uneven and irregular. True ankle-clonus does not exist in hysteria. Hysterical flaccid paralysis is *not* accompanied by hypotonus, as is organic flaccid paralysis. The reflexes in hysteria are variable, and not characteristic. Visceral, vasomotor, and trophic disturbances, hitherto grouped as hysterical, must be studied afresh. Simulation must be guarded against, and organic cases carefully eliminated. Some cases of visceral hysteria have been reported, with subsequent corrections as to the diagnosis, the discovery being afterwards made that the cases were really organic. Thus Kienboch published a case of hysterical cardiospasm, the heart being shown by a radiograph to be in a state of veritable tetanus, completely contracted, the symptoms being those of angina. Subsequent study showed the heart to be, indeed, in systole, but because diastole was mechanically prevented by a spasm of the glottis and of the abdominal muscles. If we pass to vasomotor and trophic disturbances, examples of simulation rapidly multiply, many of which are enumerated by Alquier. And many of these vasomotor and trophic cases are really examples of organic disease wrongly diagnosticated.

In conclusion, then, the physical signs of hysteria today are being rigorously revised and greatly restricted. To what extent will this restriction go? As yet, no one can say. For Babinski, hysteria cannot include that which suggestion cannot reproduce; he therefore has, for his own part, eliminated fever, trophic and vasomotor disturbances, sphincteric disturbances, modifications of the reflexes, visceral disturbances and alterations of the cerebrospinal fluid. To hysteria belong the primitive disturbances, capable of being produced by suggestion, and eliminated by persuasion; and to these Babinski has given the name "pithiatic."

What is the nature and what the cause of hysteria? Certain facts of importance seem definitely established. All who have studied hysteria have been struck by the "contradictory" character of the symptoms; for example, the absence of suffering to be expected from real paralyses, contractures, or sensory disturbances. Another striking fact is the entire absence of anatomical changes, even when painstakingly sought. These facts seem clearly to separate hysteria from organic disease, and have led numerous observers to seek the explanation of hysteria either in a functional change in the higher nerve-centers, or in a special disturbance of the psychic faculties.

Those who explain hysteria as due to functional changes in the higher nerve-centers affirm that hysteria is a special manner of reacting and of perceiving, by reason of a particular modification of the nervous dynamism, the latter being characterized by a disturbance in the cortical and subcortical reflexes. These reflexes, inhibited or excited, are the cause of the dissociation of psycho-physiologic processes, and of their isolated and indirected functioning. Thus says Raymond, as quoted by Claude, at the congress at Lausanne, already alluded to. Binswanger believes that hysterical phenomena are due to a defect in correlation between the physical and the psychic spheres; the physical irritants produce either no psychic reactions at all, or very incomplete ones; the irritation of the cortical neurones may, on the other hand, bring about abnormal reactions.

For Ziehen, hysteria is due to the extraordinary power of evolving mental representations. For example, the idea of paralysis is sufficient to evoke it. Again certain memories present themselves with the intensity of illusions or hallucinations. On the other hand, Sollier defines hysteria as a psychic cerebral disturbance, functional in character, consisting of a benumbing or a localized or generalized dormant condition, transitory or permanent, of the cortical centers, and bringing forth, according to the centers involved, vasomotor, trophic, visceral, sensory, sensory-motor and psychic manifestations.

Besides the above theories and definitions there is another set founded upon psychologic analyses. Moebius considers hysteria as due to an excess or a defect of representation. The somatic modifications of psychic origin immediately determine other modifications which are persistent. Strümpell has adopted this theory.

By his psycho-analytic method Freud has tried to restore to its old honor the popular theory of the sexual origin of hysteria. He ranges it among the protective psychoneuroses which have for a common characteristic an unconscious psychic mechanism consisting in an effort to expel from the mind an unendurable image, in painful opposition to the "ego" of the patient. Every individual reacting abnormally (by disgust) to a sexual excitation is hysterical. Hysteria is the result of a sexual desire which the subject tries to combat or to satisfy. Psychic analysis permits us to discover in each hysterical symptom, a sexual symbol; and the denials of the patient are frequently equivalent, according to Freud, to positive affirmations.

Of all the conceptions based on psycho-analysis the most interesting and the one which seems to contain the greatest amount of truth is that of Janet. He looks upon hysteria as a mental malady to be clearly outlined only by psychologic analysis. For 20 years Janet has insisted that all the phenomena of hysteria are to be explained by a limitation of the field of consciousness. The patients feel all the sensations, perform all the movements, preserve all the memories, but the consciousness does not admit them in as great numbers as in the normal state. A very large number of memories, of sensations, of movements remain in the subconsciousness; hence anesthesia, amnesia, paralysis. It is not a question of simulation since the anesthesias, as for instance the narrowing of the field of vision, are ignored by the patient, or are a matter of indifference to him. Suggestion frequently *localizes* the symptom *without producing* it. The perceptions which disappear from the field of consciousness are the least clear, and of least importance to the patient. Thus, to revert to the narrowed field of vision again, the patient concentrates his attention on the clearest visual perceptions, and leaves to his subconsciousness all those which impinge upon the less essential portion of his retina. And this very concentration of attention increases the limitation of the visual field. The limitations of the field of consciousness are variable in each individual from one moment to another, and it must be clearly understood that suggestion more easily narrows than enlarges these limitations.

Hysteria is a depressive psychosis which develops under the influence of depressing causes, such as intoxications, especially at puberty. It is very nearly related to psychasthenia, and one might almost say that hysterical subjects are merely a variety of psychasthenics. But in hysteria, unlike psychasthenia, there is no decapitation of the higher faculties. Instead the effect of hysteria is upon consciousness, upon the personality, upon the functioning of the various parts. The form of mental depression of hysteria is characterized by a tendency to the narrowing of the field of consciousness and the dissociation of the systems of images and functions, which, by their synthesis, constitute the consciousness of the individual. The narrowing of the field of consciousness leaves to the patient only a small number of ideas, which, not counterbalanced by others, easily become fixed ideas. The patients thus live in a perpetual dream. This mental state is easily distinguished from confusion and the various dementias.

Thus hysteria to-day appears to be a psychoneurosis, akin to psychasthenia. Like the psychoneuroses in general, it develops upon a soil of nervousness, all depressing causes serving to call it forth, among these moral traumata largely predominating. We do not know why these causes provoke sometimes hysteria and sometimes other psychoneuroses. That seems to be a matter of individual predisposition, at the present time unexplained. One cannot longer attribute to hysteria all the morbid phenomena which have been ascribed to it in the past, and a revision of our knowledge concerning this malady is necessary and is being made. The distinction to-day between motor, sensory and special sense disturbances of hysterical origin and organic disease are clear cut; and the study of the reflexes has considerably diminished the importance formerly attached to their modifications as signs of hysteria. Visceral vasomotor trophic disturbances and hysterical fever must be studied entirely afresh, with special reference to possible simulation, no matter how illogical the simulation may seem, or to what extreme it may seem to be carried. Suggestion must also be most carefully avoided; and above all, evidences of organic disease most carefully sought.

G. Aschaffenburg[1] attacks Freud's views of the nature of hysteria. Freud's views are best expressed by his pupil, Sadger,[2] who says: "Behind every symptom of hysteria and the obsession-neuroses is a mass of suppressed sexual desires. And in order to keep from yielding to these desires the patient prefers to remain ill, strives against a better understanding of the matter, and even renounces peace and happiness." According to Aschaffenburg, this conclusion is reached by reading into the replies of hysterical subjects more than they say or mean. This is the chief objection to Freud's idea. Freud's method of psychic analysis leads to no facts, but merely to conclusions, for most neurologists see in Freud's statements nothing more than the expression of his subjective convictions. Aschaffenburg concludes by saying: "If I should sum up my estimate of Freud's method, I would say what I have said before: it is wrong for most cases, prejudicial for many and easily dispensed with for all."

The most salient thing about hysteria is the lack of balance between irritation and reaction. Another indisputable fact is that hysteria is always found in one with a psychopathic predisposition, being an endogenous rather than an exogenous affair. A third fact is that the thing which cures is the thing in which the patient believes, no matter what it may be. Since hysteria depends on predisposition, it is well to be careful of the education of the children of the hysterical. According to Aschaffenburg, an hysterical mother usually has but one child; and this child, besides being presented with a psychopathic predisposition at birth, is all its life trained to still further misfortune by an hysterical mother. Were there more children than one they might be properly educated.

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(1) Deut. med. Woch., Oct. 31, 1907.
(2) Zentralblatt f. Nervenheilkunde, 1907, p. 45.

Symptoms. A. Gaussel[1] reports a case of *hysteria simulating paralysis agitans* in a girl 28 years of age. The patient entered the hospital with rheumatic facial paralysis, which promptly yielded to treatment. She was placed in a bed next to one occupied by a case of Parkinson's disease. The result was that she unconsciously appropriated to herself the entire symptomatology of that disease as she imperfectly observed it. Thus she presented the tremor at rest, the bent-over attitude, the feeling of heat, the stiffness, the need of moving her limbs or body, just as in paralysis agitans. Besides these symptoms, she also showed some astasia-abasia, and she walked up and down stairs without propulsion. She showed a few stigmata of hysteria such as corneal and pharyngeal insensibility, right hemianesthesia, etc.

Greidenberg published a similar but less perfect example of hysteria simulating paralysis agitans in 1888, and Rendu in 1889 reported 2 cases in which the tremor alone was simulated. Since then a few other cases have come to light. Only 1 case of astasia-abasia associated with hysterical paralysis agitans has been reported, Knapp's case in 1891, recorded in the *Journal of Nervous and Mental Diseases.*

Drenching sweats of 300 c.c. or more, occurring in hysteria in both a mother and her daughter, are reported by Hans Curschmann.[2] The mother was first affected, the sweats appearing in what she called attacks of influenza, and these were copied by the daughter. Each was firmly convinced that she *must* sweat or that she would not get well. But few other similar cases are recorded, and these are collected by Curschmann in his paper.

PSYCHASTHENIA.

The *treatment* of psychasthenia from the standpoint of the social consciousness is discussed by James J. Putnam.[3] The conscious life of any human being is interwoven with the lives of others, and it is in his social relations that the causes of the disorders that threaten his happiness

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(1) Gaz. des Hôp., Nov. 7, 1908.
(2) Muench. med. Woch., Aug. 20, 1907.
(3) Am. Jour. of the Med. Sciences, January, 1908.

and efficiency are to be sought. An individual is not limited by his skin; on the contrary, he is coëxtensive with the scope of his conscious interests and affections, and is to be considered as complete *in himself* only when his relations to others are taken into account. The social consciousness of a child is formed largely by imitation of himself and others; and with the acquisition of language and the comparison of mental conceptions, self-consciousness originates. Self-consciousness signifies the consciousness of the meaning of one's own acts and thoughts, and the recognition of good and evil. It means also the acquisition of the ability to look upon one's self through the eyes of many others. Henceforward the individual possessing it cannot live, enjoy or suffer alone, but must share his lot with others, and endure their actual or assumed reproaches. The "self-conscious" person in the morbid sense is one who has a vague idea of himself as surrounded by an imaginary circle of critics, enemies or admirers, toward whom he must adopt an attitude of shame, secrecy, hostility or superiority. "His diverse mental states may even become personified in his own eyes, so as to produce the community in miniature. He then sets himself up against himself, and, following a natural but morbid social instinct, he lends himself to the development of an inward drama, in which his varying moods, like well-trained actors, play sharply defined parts. Thus occur certain diseases due to mental dissociation in the self-conscious sphere. So, too, the depressed person intensifies his own sufferings by playing for an imaginary audience the rôle of one whose causes for suffering are greater than his own. The cure consists in showing up the real nature of the imaginary drama, proving it to be as much a comedy as a tragedy, and substituting for it the drama of real life, with its aims and incidents and motives interpreted afresh with the aid of a broader vision."

Of course much of this process takes place "subconsciously," but it is none the less the person's own waking consciousness that must in the end stand the loss or gain, according as the result comes out. Fortunately an individual cannot waver between a better type of consciousness and an inferior one without becoming dimly aware of what is going on, and aware; too, although perhaps but vaguely, of the better state which he sacrifices for the less good. "The wilfully selfish or misanthropic person is in some degree alive to the value of the better social conditions which he affects to disregard. This same principle applies to the care of the psychasthenic patients. They estimate their sufferings in part by a more or less definitely recognized contrast with a more normal state, which in reality they approve and for which they half-consciously long, and this fact is a warrant of hopefulness for their recovery, provided they are willing to be led toward a fuller realization of their ideals."

The sense of dependence upon others, which makes a person self-conscious, implies an interest in others which only needs to be developed in order to be a source of happiness and strength instead of a source of mischief and disorder. Hence in the treatment of psychasthenia due regard must be paid to the social consciousness and social environment of the patient. It surely falls within the province of education to cure these people of their excesses of individualism, which are but exaggerations of normal traits. Sometimes these traits are based upon deep-seated diseases of the brain which physician and patient alike struggle to overcome; but fortunately the reverse is also true, and of extreme importance are the cases which are now and then reported to show that tenacious delusions of suspicion and persecution may be in reality the product of narrow, barren, superstitious, savage conditions of social environment, which a broader civilization should be able to improve.

Of course there are other causes for psychasthenia than social ones, and other avenues leading to recovery than those depending upon a strengthening and purifying of social ties by means of education. This education is not urged by Putnam as a substitute for any treatment, but as a supplement to all methods of treatment. "I think," he says, "it is a distinct lack of many of the established methods of treatment that they fail to deal sufficiently with the conscious motives as means for unlocking hope and energy, and although there are many motives by which men's minds are stirred that do not point to social relationships as their cause, still the social motives are stronger than any except the religious motives, and these draw a large portion of their nourishment from the social sentiments on which they largely lean. It is not alone the claims of those that stand nearest in blood which are recognized as paramount. The demands of social loyalty are a free-mason's bond that calls out the best from college student, from soldier, from citizen, and may even inspire respect and admiration for the motives of those with whose opinions we most fundamentally disagree. * * * The "high-class" invalids who realize that by virtue of their illness they are in a position to fill a certain place of definite significance among their fellows, and the enthusiasm with which they are greeted shows that their attitude is appreciated. Such invalids were Pascal and Stevenson and many more whom every physician could name."

"Every physician knows that before he can present logical arguments to a patient for assuming such an attitude toward himself as has just been outlined, he must do something which will mitigate the feeling of emotional distress, illogical and vague, but irresistible, by which he is beset. When once this burden is relieved a little, arguments for a more rational attitude become possible. This emotional distress is multiform in character, but the most fundamental part of it is often an ill-defined yet grinding sense of social isolation which gets its primary impulse partly from without yet mainly from within. The invalid is keen to note the real indifference of those around him and is quick to magnify its meaning, quick to let it intensify the feeling that he stands alone. Sometimes the invalid's own physical weakness, his inability to conform to social standards, to compete in occupations, to take part in sports and games, raise a fence out of which his active imagination constructs a wall. He feels that his neighbors look upon him as something unlike themselves and therefore to be ostracized, and he is only too ready to justify their fancied attitude toward him and to adopt it as his own.

"Even when the sense of isolation is not felt explicitly and as such is not due to influences from without, it is often felt as an intangible cause of nameless dread. It forms a definite part of the deep sense of unreality and impairment of one's hold on the immediate present, on one's natural affections and natural responsibilities, that Janet notes as the essential feature of psychasthenia. It may be an outgrowth, too, of the sense of individualism, which if rightly understood is indispensable to sound development. It is difficult, or impossible, in tracing back these fundamental feelings, as of courage and fear, confidence and suspicion, to be sure that one can decide correctly under which long shadow the antisocial sentiment of isolation first arises. The important thing is that doctor and patient alike should recognize that it springs from various causes and may be relieved in many ways. With one person courage or confidence shown in the conduction of his own affairs may be the first step toward his elimination from the sense of isolation. With another some more definite manifestation of personal sympathy or explanation is required, especially from the physician acting as representative of the community. In other cases it is more stimulating, instead of offering sympathy, to make it clear that the community, through many of its members, stands ready to make common cause with the patient in helping him to solve the problem that confronts him, and ready to appreciate at its full value any contribution that he may make on the basis of his special experience toward the more general problem which the occurrence of sickness and pain is always urging. This is the true conception of the case. In a sense to be interpreted with discretion, but ever more generously and more reasonably, we are all members of a social 'team,' and the difficulties that beset the individual invalid are difficulties for us all.

Needs and temperaments vary, however, and so must the modes of treatment. There are many patients for whom the appeal to the highly developed social consciousness is an appeal of real power. "Such persons feel that the problem before them is not solely to get well, but also to realize that, sick or well, they are under obligations to show fidelity to leading motives which if generally adopted would develop, first opinions, then sentiments, and then traditions such as would make societies into stronger units.

Even a small increase in the sense of power derived from systematized and graduated efforts and a small increase in the sense of fellowship which comes with the knowledge that one is coöperating with others for a definite common end may effect a revolution in the life of the neuropathic invalid.

"The problem of illness is more or less like that of 'failure.' Both make demands upon our intelligence and our faith, and both may properly be regarded like the loss of a battle in a campaign which we expect to be successful in the end; if not successful in the sense of bringing victory then perhaps successful in the sense of having been the best that we could do, and if not that even, then at least of value as affording a contrast with an imagined more successful future effort of ourselves or others. It is difficult to take this attitude, and especially so when the illness is one for which we feel ourselves responsible. It is hard to live contentedly in company with the memory of one's failures, especially if traceable to one's faults, but it is absolutely essential that one should learn to do so, and very often the first step toward the relief of the torturing distress of psychasthenic symptoms is to make up one's mind to accept them as necessary companions while at the same time learning to turn as deaf an ear as may be to their words.

"Like all the pieces of self-discipline here suggested, this is to be studied like any game of skill, namely, first under the influence of the well-grounded sentiment and conviction that the end is a desirable one and the means well-planned; next, with a confidence stimulated by imitation; finally—and most important of all—under the constant binding of the new self by as strong and rich ties of association as possible to a new or wider circle of interests, or the old circle conceived in a new light; in other words, by the development of the social consciousness."

Already certain therapeutic experiments in which attention has been paid to the social consciousness have been made. It is a noteworthy and refreshing sign that in the best sanatoriums of the present day treatment by occupation to be followed by a number of patients working in company or on similar lines is counted as of much value. "A medical enterprise which secures social relationships of a special and useful sort is in operation at the Massachusetts General Hospital, where groups of outpatients are encouraged to meet regularly, under strict supervision, to compare notes as to their success in carrying out modes of treatment that had been prescribed for them, and to gain interest, information and enthusiasm for new efforts. I did not at first believe that a coöperative movement of this sort would be available for the neuropathic and psychasthenic patients, but I have changed my mind. Among those who are engaged in carrying on the experiment there are several who have been through the purgatory of nervous invalidism or are still wandering in its circles, and their experience gives them in some respects a peculiar fitness for the task."

Psychotherapy. J. Collins[1] presents some fundamental principles in the treatment of functional nervous diseases, with especial reference to psychotherapy. A diagnosis is not always necessary for the successful treatment of many neuroses, for these are sometimes cured by charlatans and "systems" innumerable. It is evident (1) that the mind has a not easily calculated influence over the body, either beneficial or prejudicial; (2) that this influence is being utilized by many persons outside the medical profession; and (3) that ignorance or neglect of the important part played by moral treatment in the cure of nervous disease tends to discredit physicians. Even organic diseases are in part controllable at times by the mind.

"The first and perhaps most important thing in the successful treatment of nervous disease is to convince the patient that his disorder is fully understood, that the features of it, which in his judgment are unique and mysterious, are perceived with a clarity that amounts almost to clairvoyance, and that the physician stands between him and danger. This may be called 'inspiring confidence,' but it is really much more than this, for the patient may have confidence in a physician's skill and judgment without harboring any of the above-mentioned convictions; nor is this relationship between the physician and the patient to be established by verbal assurance on the part of the one and the most explicit statement on the part of the other. Imaginary maladies are very rare, and it never avails to tell sufferers from them that there is nothing the matter with them, nor to treat their confidences lightly or indifferently. Indeed their fear and want of self-reliance constitute the symptoms of their very real malady."

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(1) Am. Jour. Med. Sciences, February, 1908.

The second thing is the application of the so-called psycho-analytic method to these patients. It often happens that patients with neurasthenia or psychasthenia cannot give a very definite account of their symptoms. They know they are apprehensive, agitated, anxious; that there is no peace of mind for them; but more than this they do not know. In such cases the psycho-analytic method is of great importance. "This method is based upon the association of ideas; upon the common knowledge that memory ideas are associated with sense impressions. Every one knows that an object or word seen or heard, an environment, a sound, an odor reminds us of something concrete or something which in turn brings up other associations of time, place or emotion. Psychologists have determined the conditions under which these associations arise, the influence which the frequency and the recency of the memory picture and its associated ideas have upon the kind and character of the evolved impressions as made manifest in the resulting idea. The time that elapses between the articulation of a word and the mental photograph which expresses the idea that is invoked in the hearer's mind is called the reaction time. Experimenting with a large number of words, we find that the normal reaction time for the average individual is very short, generally less than a second, and is not subject to much variation. For some words, however, if a long series of words be tried, a much longer time elapses before a verbal association is announced, oftentimes 4 seconds and more. Repetition of the test at different times shows that it is always the same words that have this protracted reaction time. If these words are selected and collated, it may not infrequently be seen that they have some personal association or reference, and, taken alone or in conjunction with the patient's story, they may be used as the key to unlock the latent memories that are, consciously or unconsciously, disturbing the patient's peace of mind. Then once they are brought up where they can be squarely attacked, they may be subjected to well-recognized modes of psychical or moral treatment."

Psychasthenia, as a disorder, originates in a neuropathic constitution which is inherited, not acquired. The chief mental symptoms are obsessions or compulsary ideas which come into consciousness abruptly and take possession of it autocratically, usurping to any degree the normal content of the mind. The patient realizes how morbid is the thought possessing him, and he can reason about it and discuss it, and in a general way vaguely analyze it, but yet he feels himself within its grasp and powerless to shake it off. A second striking feature of psychasthenia is that to which the name tic has been given. It may be simple or complex and systematized. Analogous to these systematized motor activities are the systematized emotional agitations, including morbid fears of every conceivable variety. Finally, there is an absolute lack of self-reliance. The psychasthenic hates to adopt a plan of action, he is irresolute about decision of all sorts, he is self-deprecatory, oftentimes to an absurd and apparently ludicrous degree.

The treatment of psychasthenia resolves itself into the treatment of the patient and of the heredity. The latter Collins passed over, confining himself to the treatment of the individual. The treatment of the psychasthenic is very difficult to state specifically. It entails instruction in logic, and in philosophy, given without any scholastic verbiage. It also requires the engendering of the spirit of self-reliance and courage, in order to dispel fear and doubt. "It is not sufficient to show a psychasthenic that his ideas are not founded upon facts, that his actions or conduct do not flow from logical and legitimate inferences. It is necessary first to attack the fundamental state upon which these pathological, ideational and emotional states depend. Ordinarily the most expeditious way of going about it is to restore or at least improve the general health. This is to be done by the proper employment of those measures which have been found so successful in neurasthenia: Isolation, alimentation, rest and exercise, and the use of such mechanical measures as massage, hydriatic procedure, electricity, and possibly also the use of symptom medicines. They of themselves, or the discipline which they engender, constitute the most important factor in the moral treatment of these patients, and the time when they are operative is the psychological moment to begin the moral treatment. At a definitely allotted time in the day the patient should be made to concentrate his attention upon work that requires his intellectual faculties and which is entirely foreign to self-contemplation, self-analysis and introspection. It is in this respect that, for example, appropriate literature may be of great service. The patient may be made to read, or study rather, Plato or Emerson, or Marcus Aurelius. Once the patient can concentrate his mind upon the Phædo, or such excursions in the realm of independence and optimism as Emerson's Self-reliance and Compensation, and by perhaps a veritable *tour de force* shut out his morbid ideas, the dawn of recovery is at hand. Naturally such work is not pleasurable, but neither are some, perhaps all, of the physical measures that are at the same time being utilized to restore the patient's bodily health. It is done as a task, and it should be done with the seriousness and studiousness of a task, and with the same regularity and persistency as any of the physical methods co-incidently employed. Neither is it done alone; it is done in intimate association with some competent person, or with the physician who essays to treat him. After a time the task, which seemed impossible and wholly beyond the patient's comprehension, becomes easy and pleasant, and he extends his activities volitionally in directions which are indicated to him. Naturally in the beginning the pathological ideas assert themselves, and the physician is likely to be impressed with the aboulia and distractibility of his patient, but there are few cases that resist confident and protracted effort along these combined lines. Gradually there is brought about a reëducation of will power, a keenness and intensification of intelligence, and an expansion of sentiment. The patient's resources are increased. Life begins to have some charm for him, existence some purpose, and, with the aid of the staff which the physician puts in his hands to steady him over trying and difficult stations, the patient is restored to a useful life, often to distinction and eminence."

ANXIETY NEUROSES.

In the Practical Medicine Series, Vol. X, 1903, p. 138, a case of akathisia, as described by its discoverer Haskovec, was reported, and 2 illustrations were presented with the summary. Since then cases have been reported by Raymond and Janet, by Bechterew and Beduschi. The essential phenomenon in akathisia consists in the impossibility of remaining in a sitting posture, on account of the appearance of transitory psychic or somatic nervous symptoms, chiefly vaso-motor. In nature it is an anxiety neurosis. As soon as the patient changes his attitude all the symptoms at once disappear. Raymond and Janet have observed that there is an hysterical form of akathisia in which the patient apparently forgets how to coördinate his muscular movements in sitting, this form being properly called *akathisia paralytica.* Sometimes the act of sitting down brings on convulsive phenomena, this being called *akathisia spastica.* The most frequent form, *akathisia psychasthenica,* is the one in which the seated posture brings with it anxiety, general suffering, alterations in the breathing, heart-beat and acceleration of secretion of perspiration. To this category belong the 2 cases of Haskovec, the 2 cases of Beduschi and the case of Raymond and Janet.

A. Stcherback, of Warsaw,[1] now reports an alleged case, in a boy of 12 years of age, with neuropathic heredity, very impressionable, and suffering with sick headaches. From 5 to 15 minutes after seating himself, he felt a disagreeable sensation in the lower part of the thigh and upper two-thirds of the calf of the left leg. This sensation augmented and forced him to stretch out the leg, and then came the tendency to stand up. As soon as he stood the disagreeable sensation left. There were no convulsive phenomena, trembling, anxiety, etc., as in other cases of akathisia. To this condition, cured by suggestion, the author gives the name *akathisia paresthetica.* The im-

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(1) Le Progrès Med., May 30, 1908.

pulse to get up was so pronounced that at school the boy had permission to stand every 5 to 10 minutes during school hours. The psychic state of the patient was observed to have much to do with the intensity of the symptom, it almost disappearing when the mind was absorbed about anything.

NEURASTHENIA.

Blood-pressure in Neurasthenia. E. D. Macnamara[1] has investigated the blood-pressure in neurasthenic states and the effects of certain forms of treatment thereon, and arrives at the following conclusions:

1. In many cases of neurasthenia there is an alteration of the level of the blood pressure from the normal, the level being sometimes higher and sometimes lower than normal.
2. Patients who show such alterations and who undergo certain sorts of treatment manifest, in some cases, at the end of a course of treatment a level of pressure different from that which existed before treatment began. If the pressure at the commencement be abnormally high it will probably descend, while if it be abnormally low it will probably ascend.
3. The number of patients whose blood-pressure is different at the end of their course of treatment from that which it was before treatment began is greater among those without a family history of nervous instability than among those with such a history.
4. In a very large majority of cases the application of high-frequency currents produces a lowering of blood-pressure, while the static bath (+ charge) and massage produce a raising of pressure. The discharging of a patient charged with static electricity results as often in a rise as in a fall of blood-pressure. The application of the faradic current tends rather to lower than to raise the blood-pressure and the galvanic current cannot be said to effect much change in either direction.
5. It is difficult to institute treatment with any confidence that there will be at the end of the course such an alteration as might perhaps have been expected from a

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(1) The Lancet, July 18, 1908.

knowledge of the alteration that is likely to follow each application of the therapeutic agent employed, though we may look for a change of level, when the level was at the commencement abnormal, to one that is nearer the normal.

6. There is no evidence that the differences of variation of level before and after treatment which may sometimes be noted can be correlated with improvement in health.

Treatment. R. Bing[1] discusses the principles and indications in the medicinal and dietetic treatment of neurasthenic individuals. After mentioning the danger of routine schematic treatment for all cases, he divides the symptoms of neurasthenics into 3 groups: (1) the psychic symptoms, for which psychotherapy is necessary; (2) the subjective disturbances, such as headache and backache, insomnia, anorexia, vertigo, etc., for which psychotherapy is again first to be made use of, and medicines more for the sake of suggestion than for physiologic effects; (3) the objective symptoms, such as increase of the tendon reflexes, increased irritability of both muscles and nerves, urticaria factitia, dermographia, attacks of tachycardia, abnormal and widely varying pulse-rate and pulse-pressure, tremor of the hands and fingers, hyperidrosis and abnormalities of secretion on the part of the stomach and intestines; for these drugs and dietetic treatment are indicated.

For the latter group of symptoms bromids are almost universally used, but they never give any results, if indeed they do not actually harm in some instances; for some neurasthenics, as Oppenheim observes, have an increased susceptibility for bromids. These persons are exhausted and depressed psychically and physically, and the appetite and digestion are apt to be influenced unfavorably. Bromids should be given only when irritative conditions dominate the clinical picture, as motor unrest, trembling, palpitation, jactitation at night, or anxiety-neuroses, in which case 45 grains a day may be given, very well diluted in water.

Codein has been recommended as a substitute for sodic bromid, and is especially useful in the mild melancholia of neurasthenia at times present. It should be given 3 to 6 times daily in doses of 1/6 to 1/4 grain. Though Donblüth and Freund are enthusiastic about the codein cure, Bing shares Erb's skepticism as to its great value, though Bing admits that in many cases it is an efficient remedy. It is sometimes well to prescribe a mixture of sodic bromid and codein phosphate.

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(1) Therap. Monatshefte, July, 1908.

A third sedative is cannabis Indica, given in very small doses over a long period of time, in the form of pills, alone or with other drugs. Bing recommends

℞	Quininæ sulphatis	1.0
	Acidi arseniosi	0.06—0.10
	Ext. cann. Ind	0.45
	Ext. et Pulv. rad. Valerianæ, q. s. ut fiant pil. No. xxx	

Sig.—One pill each evening.

Among the tonics, arsenic stands high, especially in the emaciated, exhausted, and depressed neurasthenics. The dose used should be very small, not more than 3 to 5 drops of Fowler's solution or 1/30 to 1/40 of a grain of arsenious acid *per diem,* and the arsenic should be continued for weeks and months. Cacodylate of soda in ¼-grain doses may be given if the patient is unable to take other forms of arsenic. If anemia is present, iron should be given. Erb's favorite prescription is:

℞	Ferri lactici,	
	Quininæ sulph	āā 4.0
	Ext. nuc. vom	1.0
	Ext. Gentianæ q. s. ut f. Pil. C.	

Sig.—One to two pills after each meal.

A drug combining both tonic and sedative characteristics is valerian, which is a most reliable agent for overcoming vasomotor and cardiac disturbances. With its use the arrhythimia ceases, the pulse slows down, the heart-action becomes stronger, the blood-pressure steady. Bing uses valerian tea. In the morning a heaped-up teaspoonful of the root is placed in a large cup of cold water, and allowed to stand still till night; with occasional stirring. This is then filtered through linen, and taken. Another cupful is started at night in the same manner, and is taken the next morning. This treatment is carried on for months.

If prepared with hot water the tea is weaker, since important constituents are vaporized and lost. The taste is bad, and pills may have to be used in accordance with the following formula:

℞ Ext. Hyoscyami,
Zinci oxidi................................āā 5.0
Ext. valerianæ................................10.0
M. F. pil. No. C.
Sig.—One or two pills three times daily.

Phosphorus preparations are indicated in secretory neuroses, as nervous dyspepsia, or in cases of emaciated individuals who are at the same time anemic. Bing prefers sodic phosphate, dissolved in water, a half teaspoonful after each meal. Glycerophosphate of calcium is also useful. This is a powder easily soluble in water, and is given in doses of 3 or 4 grains four times daily. If the sodic phosphate has too laxative an effect, calcic glycerophosphate should be given instead. The expensive organic phosphorus preparations are not recommended.

As to general treatment, it should be insisted upon that the patient take some little nourishment at least every hour, and never permit his stomach to be empty. A glass of milk, a piece of milk-chocolate, a cooky, cracker or a fig, or something of the sort, should be on hand, ready for the neurasthenic every hour. Of these, milk is by far the best thing to be taken, and if distasteful, can be rendered less so by adding tea to it, or salt, sodic phosphate or bicarbonate, or by the use of buttermilk. Eggs, milk, and vegetables form an excellent general diet for these cases, all irritating foods being carefully avoided. Tobacco and strong coffee must often be forbidden. Alcohol is injurious, but a glass or, at the most, two glasses of beer may be permitted each day. In general, it may be said that each case of neurasthenia is a study in itself, no two cases being alike. Therefore no two cases can be treated alike.

A. L. Pringle[1] advocates the use of hydrotherapy and electricity in the treatment of neurasthenia. A great deal may be accomplished at home by a judicious use of salt-water bathing at various temperatures, together with mas-

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(1) Edinburgh Med. Jour., October, 1907.

sage. Neurasthenics likely to be benefited by hydrotherapy are those whose condition is dependent upon (1) toxic states, (2) fatigue, either from business worries or home worries, and (3) shock, mental or physical. Most cases fall in group 1, and in them there is always some form of digestive disturbance, either gastric or intestinal. There is an associated condition of flatulence and constipation, with frequently the passage of mucous shreds. Such cases are helped by drinking natural sulphur waters, for these will thoroughly wash out the whole alimentary canal and rid it of toxins. Often the following treatment is of advantage: "The lower bowel is washed out by means of a hydrostatic douche connected with a soft rectal tube, the pressure, temperature, amount and variety of mineral water employed being at the discretion of the medical attendant. Usually 2 separate injections of from 20 to 60 ounces or more are employed. Subsequently the patient is placed in an ordinary immersion bath, the temperature of which is kept at about body heat. Beneath the surface of the water is a flexible pipe terminating in a finely perforated rose, through which a hot douche at any required temperature can be played over the abdomen. The general condition of the patient is later on toned up by the various forms of thermo-mechanical baths, such as the needle, Harrogate douche, Aix or Vichy massage douche, by which the functional activity of the vasomotor fibres is stimulated, and the blood-pressure raised or lowered according to the variety of the bath employed. The patient is enjoined to rest as much as possible, the greater portion of the day being spent, if possible, in the open air. Frequently the heart in these cases gives considerable trouble; sharing in the general fatigue, it gives rise to symptoms of faintness and palpitation. The Nauheim and Schwalbach baths may be employed here with marked success.

"In the third group of cases which are ascribed to shock, the nervous system must be soothed. This is accomplished by means of hot sulphur immersion baths, peat or mud baths, and at the same time the general condition of the alimentary canal must not be overlooked. As improvement takes place, the baths may be of a more stimulating variety, till at length the patient is able to take the full benefit of the Aix or Vichy massage douche." As to electricity the author inclines toward high frequency and static electricity.

E. Régis[1] discussed the question as to whether neurasthenics should be sent to the seaside or not, before the congress of climatotherapy at Biarritz in 1908. The answer to this question depends on the psychic and somatic idiosyncrasy. The psychic element plays a large part in the treatment of any patient suffering from any ailment; but in neurasthenia it plays an especially prominent part. If a neurasthenic has a taste for the sea and loves sea-bathing, he will do very well; but if he dislikes the sea, he will surely be harmed by being sent to it. The sea has a wonderful influence over some; among certain ones it evokes a feeling of sadness, a sense of vastness and isolation, of lonesomeness, homesickness, and melancholy. To others it brings a feeling of rest, of contentment, or of exaltation of body and mind. The neurasthenic must be studied from the point of view of the possible psychic effect the sea may have upon him. His body, too, must be considered. Some cannot well support the climate. Ordinarily body and mind go together, but sometimes they do not. Usually it is thought that the sea is of value to the depressed and is harmful for the excited, but Régis does not share this general, almost universal, opinion. Indeed, excited states sometimes quickly yield at the seashore; provided, of course, that they are not associated with any organic lesions of the central nervous system.

Régis deduces the following conclusions: (1) One of the principal indications for ocean climate in neurasthenic states is to be found in the individual syncrasy, whether psychic or somatic. One may with advantage prescribe that climate for neurasthenics who have a taste for it, are attracted by the sea and impressed and cheered up by sight of the sea. Those who do not like the sea should be kept away from it, as well as those whom it renders sad or melancholiac. (2) It is still generally admitted that a sea climate is for the depressed, but not for the excitable. The reverse is the case. It is certain that the excited neuropaths grow calm at the seaside, and that the depressed are sometimes excited by it. (3) Except in cases of grave intolerance, both infantile and juvenile neurasthenic patients improve notably at the seashore. Almost all sorts of neurasthenic women are especially helped. An associated hysteria does not constitute an obstacle. (4) The indications of seaside treatment do not depend upon the cause of the neurasthenia. (5) No matter what the clinical form of the neurasthenia, it may be aided by sea-therapy.

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(1) Jour. de Méd. de Bordeaux, April 12, 1908.

EPILEPSY.

* **Diagnosis.** G. Paul-Boncour[1] discusses the question of the diagnosis of epilepsy when no convulsions exist. It has been affirmed by some that certain intractable children, not having convulsions are, notwithstanding, cases of epilepsy, and so the "epileptic character" has been described. This is said to be essentially indicated by an exaggerated excitability betraying itself by abrupt changes in emotional tone, and by sudden and violent impulses. The young epileptic is taciturn, egoistic, rancorous, choleric, headstrong, quarrelsome, deceitful. He is hard to get along with. He laughs and cries easily, has no manners, is bereft of ideas, except those of destroying or injuring, and of torturing animals and playmates. Perpetually in motion, he is capricious; and often life with him in the family becomes impossible. Now, if this "epileptic character" really exists, ought not one to be able to make a diagnosis of epilepsy on the basis of the above characteristics? Assuredly, no one should do so; for all of these characteristics are to be found in the merely unstable school-boy, or in the neuropath. Instead, then, of the term "epileptic character," the term "character in epileptics" should be used, for the former heading will lead to a false diagnosis of epilepsy where epilepsy does not exist.

Edouard M. Dupaquier[2] gives something of a summary of Paul-Boncour's article on the "epileptic character," quoting his article in places, as it appeared in *Le Progrès Medical*. Dupaquier agrees that there is no such thing as

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(1) Le Progrès Méd., Dec. 21, 1907.
(2) New Orleans Med. and Surg. Jour., March, 1908.

diagnosing the epileptic character in the interparoxysmal period. Really all that is meant by saying a child has the "epileptic character" is that he is unstable and neuropathic. He certainly is not an epileptic because of such instability and nervousness.

Traumatic Epilepsy. E. D. Martin[1] discusses traumatic epilepsy and reports 2 cases, both of the Jacksonian type. He takes the view that many so-called cases of idiopathic epilepsy are really traumatic, and reports a case so diagnosed. Careful examination, however, showed that it was the Jacksonian variety. It occurred in a boy whose whole nature was changed. He was absolutely unmanageable, without a particle of judgment or reason. While awake he was constantly moving about, and sometimes would repeat the same word or sentence continuously. Operation practically cured him, in that he has had only 2 convulsions since. The operation was done in March, 1903, and the last communication from the patient was in May, 1907. Though the boy's mother had given to the physicians and the specialist whom she had consulted the history of a heavy object falling and striking the boy on the parietal eminence on the left side, the information was uniformly disregarded or set at naught. Yet careful observation showed the convulsive movements to begin in the face, hands, and arms, and then continuing to the lower extremity of the left side, before the attack became general.

Late Epilepsy forms the title of a paper by L. Marchand and H. Nouet.[2] By this they understand epilepsy developing after 30 years of age, but they separate from all these cases those occurring after 50 years of age, to which the term senile epilepsy is usually and very properly applied. The etiology of late epilepsy seems to be entirely different from that of idiopathic early epilepsy. According to Fürnster late epilepsy is caused by trauma and old cases of encephalitis, sometimes dating back to childhood. In some instances heredity is undoubtedly a factor. Arteriosclerosis is sometimes found. Senile epilepsy has been ascribed to arteriosclerosis, syphilis, alcoholism, diffuse or-

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(1) New Orleans Med. and Surg. Jour., March, 1908.
(2) Gaz. des Hôp., Sept. 19, 1907.

ganic lesions of the brain and its membranes, and heredity. The crises, often accompanied by vertigo, precardial anxiety, and insomnia, are just like those of idiopathic epilepsy.

The authors report 2 cases of late epilepsy, the first in a young man, 25 years of age when he had his first convulsion, and who showed at his death in his thirty-seventh year no visible gross cerebral changes, but microscopically a diffuse sclerosis localized in the molecular layer of the cerebral cortex; the second in a woman, 50 years of age at her first convulsion, who showed at her death in her fifty-eighth year, no atheroma in the basal arteries, but small foci of softening in the gray nuclei of the 2 hemispheres, some disseminated fibrous thickening in the meninges, and a neuroglial sclerosis in the superficial area of the cortex, in the most peripheral part of the molecular layer.

Many cases of late epilepsy occur in the insane, first in those showing the psychic equivalents years before the convulsions appear, and secondly in other insane patients apparently not epileptic. Thus in dementia precox, senile dementia, and circular insanity, epileptic crises occur. In these cases there will be found chronic lesions of the meninges and scleroses of the superficial portion of the cortex. To this category belongs also general paralysis of the insane, the crises being the apparent result of the diffuse meningo-encephalitis present in these cases.

According to the authors, late epilepsy differs in no essential respect from ordinary idiopathic epilepsy of the young, except in the time at which it develops.

Nocturnal Petit Mal. L. Pierce Clark insists upon the curability of a rare form of nocturnal petit mal by the use of large doses of bromid.[1] Rapidly recurring nocturnal petit mal is a rare form of sleep epilepsy occurring both independently of, and in connection with, grand mal seizures. The general exhibition of the type is the same, no matter whether it is associated with grand mal or not. Bromids, as ordinarily given, invariably increase the attacks, so that many observers have classified these sleep disorders as hysteria, pavor nocturnus, somnambulism, and the like. Clark says he has seen but 4 cases of this type

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(1) **Am. Jour. of Med. Sciences, January, 1908.**

of epilepsy among several thousand cases. Aside from the peculiarity of the attack itself, this sort of epilepsy does not differ in cause or course from idiopathic grand mal.

"The attacks of nocturnal petit mal invariably occur while the patient sleeps, either by day or night. Usually, as soon as the patient falls asleep the attacks begin. There may be as many as 300 separate and distinct attacks in a single night. The patients usually awaken from deep sleep, the eyes are opened widely; the pupils are dilated and irresponsive to light, the head is moved from side to side. There is an anxious and furtive look in the face. The face may be congested or very pale, and is usually the one or the other to an extreme degree. In a few seconds after the onset the patient executes some incredibly rapid movements of the hands or feet, but with no clear intent or purpose, such as is seen in somnambulistic states. Sometimes patients exhibit attention when spoken to, but they make no coherent reply. Unless disturbed, the patient passes into normal sleep at the end of the attack."

Clark reports all 4 of his cases, of which the first may be taken as a type. This patient had as many as 200 similar attacks during a night. Clark made the following notes of this case: "The patient suffered a mild attack at 9:20 a. m. (He had retired at 8:30 a. m., and was sleeping by day.) The patient was rather restless in his sleep; occasionally he opened and closed the hand and twitched the feet. At the onset of the attack he opened his eyes in a wild, frightened manner, and lay quietly for about 5 seconds; the pupils dilated widely, and upon application of the light test the patient was startled and closed the eyes several times, but the pupils remained irresponsive to light. He then sat up in bed, clutching the mattress. He flexed and extended the legs rapidly. The lips were drawn back from the teeth, and the teeth were tightly closed in a demoniacal grin; he slowly turned his head from side to side several times. The attack ended in perfect and instantaneous consciousness. The pupils readily responded to light. The whole phenomenon had lasted but 30 seconds. Five minutes prior to the attack his pulse and respiration were normal, but during the attack his respiration was rapid and very heavy, almost stertorous; and 1 minute after the attack his pulse was 94 and respiration 22. The face, which was highly congested during the attack, remained somewhat flushed after the seizures. The patient had but 2 typical grand mal epileptic attacks in as many years."

Important is the method of treatment by bromids. "Bromids in ordinary dosage (60 to 120 grains daily) were not only ineffectual in controlling the attacks, but actually increased the number of paroxysms threefold over that of the non-sedation period. However, on a daily dosage of 130 to 230 grains the seizures became less frequent and severe, and finally ceased altogether after 5 days' treatment at 400 grains daily. The high-dosage experiments finally showed the sedative level was 230 grains, at which amount medication was sustained for several months. The patient had no more awakenings nor any other epileptic manifestation whatever, and he is now in excellent physical and mental health, which is in marked contrast to his former hopeless state.

"The principle of high dosage of bromid may be stated as a form of hyperbromidism, bromid intoxication, or poisoning. The drug is given in a steadily-increasing dose, as that of iodid in specific disease. The patient has high rectal irrigation once or twice daily to reduce the amount of congestive catarrh, eliminate toxins induced by faulty metabolism or deficient oxidation, and for its diuretic effect. The patient is also given prolonged hot packs and baths at night for diaphoretic effect, and cold douches, sprays, and cold drip-sheet treatment mornings to stimulate the peripheral sensory nerves and to preserve vasomotor tone. Vigorous deep massage is steadily maintained to cleanse the muscular circulation.

Treatment of Idiopathic Epilepsy. A. J. McCallum,[1] in charge of an epileptic school for boys, makes a plea for the early use of large doses of bromids. In discussing treatment he says:

"Epilepsy being due to severe, prolonged, or repeated sensory simulation of the brain, I use that amount of bromid that is necessary to stop the sensory stimulation. The

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(1) Brit. Med. Jour., March 14, 1908.

amount required to do this varies exceedingly. Eighty grains a day is the average, but so small a dose as 60 grains has been sufficient to arrest epilepsy of an intensity that was to be estimated by the occurrence of 267 'grand mal' seizures a year. Among the boys at the colony now, 130 grains a day has been the highest dose given so far; but I have given 320 in private practice and arrested the disease. The method is to give 20 grains night and morning, and increase by 10 grains per day as often as fits recur. Epileptic boys stand bromid very well, 80 or 100 grains per day are taken without their showing any signs that they are taking a drug; 150 or 200 grains per day begins to interfere with the equilibrium—their walking becomes uncertain, and they ought to be kept in bed. With larger doses, swallowing and organic reflexes are interfered with. Adults do not stand bromid nearly so well. The boy that had 320 grains had sordes on his lips and gums, his breath was foul, swallowing was difficult, there was slight hematemesis and melena, respiration was shallow, and râles were heard. The fits, which were numbering 8 and 10 a day, stopped, the bromid was gradually reduced, and the boy had no more fits for 2 years.

Respiratory exercises and *fenestration of the skull* are 2 methods of treatment for epilepsy advocated by William Alexander.[1] He believes that the intense cyanosis observed in the attacks is beneficent in character, that it is the antidote which arrests the spasms. In some cases the cyanosis has to be extreme to relieve the spasm; in other cases the cyanosis need not be very marked. Indeed in petit mal the respiratory pause alone suffices to check the attack. Alexander believes from his observations in operating upon epileptics, that in epilepsy there is, just before the fit, an excess of clear flaky fluid in the meshes of the arachno-pia, and that the veins are at the same time small in diameter. By holding the breath these veins are congested and the fluid is driven away. Experiments were tried upon such epileptics as had the intelligence and control necessary to hold the breath, and the breath was held by these patients from 60 to 125 seconds at a time for 3 or 4 times at a session. In 9 cases the attacks in-

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(1) Liverpool Medico-Chirurgical Jour., January, 1908.

creased under treatment, in 7 cases the attacks remained the same in number, and in 19 the attacks markedly diminished; these 19 at the time of the experiments not having received any medicine.

Alexander treats certain intractable cases by fenestration of the skull, removing a piece of bone over the motor cortex, about 4 by 3 inches in size. His first case was reported by Dr. Carter in the *British Medical Journal,* February 9, 1907, and though the patient had 80 seizures the day before the operation, at which a piece of bone 2½ by 4½ inches was removed from over the right Rolandic area, she never had a fit after the operation. The dura was not incised in this case. Eight other cases are also reported of a similar sort. The operation is attended with little shock and no serious hemorrhage.

J. Courmont and Crémieu[1] have carefully studied the effects of *dechlorization* in an epileptic treated with large doses of bromids. In 1904 J. Voisin, R. Voisin, and L. Krantz reported as a bad effect of dechlorization, mental troubles, chiefly melancholia, depressed states, mental confusion, and hallucinations. The melancholia was quite unlike an epileptic delirium, for the patients remembered it afterwards. Neither was it due to the mere effects of bromism, for there was no abolition of the pharyngeal or pupillary reflexes, acne, fetid breath, and the other usual signs of bromism. Weight was maintained, so that the mental disturbance was not due to starvation. Experiments made the following year by the Voisins and Rendu led to the conclusion that nothing noteworthy is seen if one merely deprives of his salt an epileptic taking no bromid; but that if one deprives of salt an epileptic absorbing 4 grams of sodic bromid (61.7 grains) per day, severe disturbances may follow, even leading to a fatal issue. At all events, 4 grams of bromids constitute a toxic dose in an epileptic deprived of salt.

The case studied by Courmont and Crémieu was very carefully observed, and a chart was prepared, showing the patient's general condition during a little over 3 months' time, the number of seizures while on a dosage of 2.5 grams of bromid, without dechlorization, and the onset and dura-

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(1) Lyon Méd., June 28, 1908.

tion of the mental disturbances while on a dosage of 4 grams with dechlorization. There was a complete cessation of the seizures for 35 days, the last one occurring 3 days after the beginning of dechlorization, and the next one following occurring 15 days after its abandonment. The mental disturbances began 15 days after the beginning of dechlorization and the administration of 4 grams of bromids daily, and lasted 10 days after the treatment had been abandoned. The author's conclusions, confirming our present knowledge, are as follows: The daily administration of 4 grams of potassic bromid to an epileptic subjected to a complete or almost complete dechlorization, may be followed in a few days: (1) by a disappearance of the seizures, (2) by a grave nervous condition, with excitement, attempts at suicide, and even by a fatal issue. This method is therefore to be used only with extreme prudence, and salt must be given the patient as soon as untoward symptoms develop. A dose of 1.5 grams of bromid is sufficient during dechlorization.

CHOREA.

Symptomatology of Chorea Minor. F. Langmead[1] complains of the lack of attention which "has apparently hitherto been paid to the changes in the movements of the iris muscles which are frequently to be seen in chorea." Most of the text-books give extremely meager accounts of the eye-symptoms of Sydenham's chorea, which the author enumerates as follows:

"1. *Hippus.*—The first is hippus. In many children who are more than slightly affected by the ordinary sthenic type of chorea the movements of the iris are extraordinarily wide and rapid. This is readily explained by the jerky movements of the eyeballs and the consequent quick and frequent variations in the amount of light which reaches the retinæ, and also the suddenly altering accommodation for the many objects which in turn are included in the visual field. This necessarily renders the detection of rhythmical oscillatory movements of the iris no easy matter, but during intervals of quiet these movements, which constitute hippus, are sometimes noticeable.

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(1) The Lancet, Jan. 18, 1908.

"2. *Peculiarities of Movement of Accommodation.*—That the contraction of the pupils to accommodation may be extremely rapid and sudden has already been mentioned, but it will frequently be seen also, that the reaction is asynchronous on the two sides, one pupil contracting, while the other remains temporarily dilated. This is especially marked when the pupils are unequal, the larger usually reacting more slowly.

"3. *Contraction.*—Contraction of the pupils both to accommodation and to light is usually ill-sustained, and here again the affection may be unequal, so that when both are contracted one will sometimes be seen, as it were, to tire out and dilate whilst the other remains small.

"4. *Varying Inequality of the Pupils.*—It may be noticed in some cases that one pupil remains persistently smaller than the other during the complete examination. This may continue for several weeks and only be replaced by the normal equality when the child has recovered or, on the other hand, when next seen the pupils may be equal or that which was formerly the larger may now be the smaller.

"5. *Eccentric Pupils.*—Eccentricity of the pupils may occasionally be present and may become better marked when the pupil is contracted and less obvious when dilated.

"As far as I have been able to judge, it would appear that these phenomena are by no means uncommon and bear no relation to any particular form of chorea. The pupil is no more often or obviously affected on the side on which there is greater movement, or paralysis, than on the other. Mere inequality of the pupils is of little significance, for it is commonly found among children, but I have not found the hippus, the altered accommodation, or the eccentricity of the pupils, in any other general condition, except articular or cardiac rheumatism. This is of slight interest from the point of view of the common etiology of rheumatism and chorea."

* **Treatment of Chorea Minor.** A. F. Voelcker[1] discusses

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(1) **Buffalo Med. Jour., June, 1908, and Folia Therapeutica, April, 1908.**

the treatment of chorea which he divides into 3 heads: (1) general; (2) symptomatic; and (3) specific.

"(1) *General.*—Directly a case of chorea is recognized the child should be taken from school, or if at home should be kept apart from the other children in the house, at all events for the greater part of the day. Leniency must be shown to the shortcomings of children, and particular care taken that the children are as far as possible kept away from those environments which have in them the makings of a difference of opinion or of rivalry of interest. At this stage much harm may be done by injudicious parents, foolish nurses, or teasing brothers or sisters. Isolation in the early stages is undesirable, if by isolation we mean shutting a child up by itself. Rest is a very important requirement at this stage, but note must be taken of the temperament of the child, for in some cases the forcible detention of a child in bed all day long will do more harm than good. On the other hand, it is essential that the child should be made to lie down for at least part of the day, though there is no reason why this procedure should be made tedious or punitive by deprivation of toys, books, or of mechanical work. Sewing or knitting should only be allowed when those arts have been already acquired, and new kinds of work should not be attempted. At this period inattention and instability, the two most prominent features of the condition, are to be met, not by correction, but by judicious sympathy.

"The question of exercise must be largely determined by the coexistence of other rheumatic manifestations, as indicated by the temperature, by the state of the heart, and by the presence of pain. In the absence of these there is no harm in letting the child go out for short walks, but it must be remembered that any fright, over-fatigue, or excitement may have the result of causing a rapid development of symptoms.

"By the adoption of these general hygienic precautions and by feeding the child well and securing sleep by means of a warm bath at night, and by the administration of sedatives, I think it is quite possible to avert an attack of chorea. For the purpose of securing sleep I have found nothing so useful as the administration of trional in 5-grain doses every 6 hours.

"(2) *Symptomatic.*—The symptomatic treatment of chorea will in the main resolve itself into the treatment of the movements, insomnia, the emotional upsets, the wasting, and of the paralytic phenomena which may constitute the great feature of an attack of chorea.

"*Movements.*—Directly these are marked the child should be put to bed and kept there. The bed should not be too large and should be protected at the side, so that there is no risk of the child falling out, and if the movements are at all severe it will be necessary to have the sides of the bed padded. In the worst cases of movement it may be necessary to make the bed up on the floor, but this is a measure which has great disadvantages, as it increases the difficulty of nursing the child, and is not without risks from the liability to draught and dust, while the unusual situation is not particularly reassuring to an excitable child." Bed sores are fortunately very rare. If movements greatly distress the child, they may be controlled in part by wrapping in a blanket and then tucking the patient firmly into bed. Avoid the appearance of punishing the child.

"Choreic movements should not be allowed to go on unrestrained. The measures we may employ to diminish the movements may be divided into external applications and internal medication. (I) *External applications* include packs, baths, douches, sponging, massage, and electricity. Of all the external agents there is none so satisfactory as the use of the hot-pack. The child is wrapped in a blanket, which is wrung out in hot water, covered over with a mackintosh sheet, and then with another dry blanket, and thus enveloped is tucked up in bed. In such a warm pack a child may be left for several hours. Cold-packs are indicated when the case is complicated with severe pyrexia, but apart from that they do not offer any advantages over the hot-packs. Cold douches and tepid sponging find their chief utility during the period of convalescence or in very chronic cases. Blisters and counter-irritation along the spine need only to be mentioned to be condemned. Massage is undoubtedly of great benefit when the movements are subsiding, but has its greatest utility in the treatment of the wasting and of the loss of power after prolonged chorea, either of the motor or the paralytic variety. Electrical treatment may be useful under similar circumstances," but simple massage is easier and better.

(II) *Internal Medication.* Arsenic sometimes helps but often fails. The author finds no benefit from its use in the acute stages of chorea, though it is useful in combination with iron and nux vomica in convalescence and in the paralytic forms. Ergot alone or with strychnin has been found wanting.

(3) *Specific* treatment has been based upon the theory of the rheumatic origin of chorea, and salicylates and aspirin have been recommended. In general salicylates are of no value, except for associated rheumatic conditions, such as tonsilitis, arthritis, endocardial and pericardial lesions, and pyrexia. The author has therefore abandoned salicylates in uncomplicated chorea.

In uncomplicated cases he favors the administration of sedatives, chiefly bromids, chloral, and chlorotone. The first two of these he dislikes, particularly chloral, but the latter has brought with its use very gratifying results. The period of the attack, the severity of the movements, and the mental instability are all favorably influenced by it. It has some minor disadvantages, such as making the children drowsy, and there is sometimes produced an erythematous rash with puffiness of the eyelids, though there is no albuminuria. Better than all of these drugs is trional.

"Trional is practically insoluble in water, so that it is best administered in the form of a cachet or else in "tabloid" form, or given in suspension. Given in these forms the drug does not cause gastric disturbance, vomiting is not observed, nor does the appetite suffer. The following mode of exhibiting trional has been suggested:

℞	Trional	gr. xv.
	P. sacc. alb	ʒii.
	Gum. trag.	gr. iii.
	Misce. Fiat. emuls.	
	Gum. arab.	gr. iii.
	Aq. flor. aurant	ʒii.ss.
	Aq. laur. ceras	ʒss.

One-third part to be taken in milk or water as a single dose.

"The dose of trional with which I generally commence is 5 grains 3 times a day for a child over 4, but this dose should be increased by more frequent administration, so that the child is taking 5 grains every 6 or every 4 hours. It is, I believe, more advantageous to administer the smaller doses at shorter intervals than to give larger doses at longer intervals.

"When choreic movements are subsiding the dose of trional should be diminished by reducing the frequency of administration, and during convalescence preparations of iron, arsenic, and nux vomica, or cod-liver oil and iron, will prove most useful. Headache, which is a not infrequent accompaniment of chorea, is best treated by aspirin or by a combination of phenacetin and citrate of caffein. For maniacal conditions the hot-pack is of the greatest use, while in hyperpyrexia the use of the cold pack or cold bath is imperative.

"When we meet with concurrent chorea and arthritis, tonsilitis, pyrexia, or peri-, myo-, or endocarditis, then it is best to administer both trional and an anti-rheumatic drug, and for this purpose in children I prefer aspirin or salol to the salicylate of soda. In cases of pericarditis, the application of an ointment containing 1 dram of oil of gaultheria to 1 ounce of lanolin over the precordium will often give great relief."

The treatment of chorea minor, with especial reference to the danger of arsenic therapy, is discussed by Henry Koplik.[1] "It has been my custom in all cases of chorea to watch the action of the kidneys, more especially in cases which are receiving the arsenic therapy. My method has been to begin with 1 drop 3 times daily of Fowler's solution and to increase *1 drop* a day (not 3 times daily) until a point has been reached which is called tolerance; that is, until there is a slight puffiness of the eyelids. As a rule, with older children 10 drops 3 times daily is a maximum dose. A close study of the urine revealed a most interesting phenomenon. In some cases before any stomach uneasiness or puffiness of the eyelids were apparent a slight trace of albumin appeared in the urine. If the administration of the drug was continued casts

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(1) **Medical Record, Jan. 18, 1908.**

would appear, and in some cases blood cells. At first the cases were regarded as exceptional." It was soon found, however, that such cases are not exceptional, but that a large proportion of cases so reacted to arsenic. Even 5 drops 3 times daily has caused albuminuria. "We have proven to our satisfaction that in a definite number of cases Fowler's solution is an irritant to the kidney, causing the excretion first of a trace of albumin, which in time becomes more marked, and is then accompanied by casts and blood cells. In view of the above, long before a puffiness or edema of the eyelids occurs, we have in a study of the urine a most delicate test of the point of tolerance for arsenic. That is, when a trace of albumin occurs in the urine it is time to stop the drug no matter how small the dose or how excellent the general condition as to the other signs, such as the absence of puffiness about the eyes or gastralgia. So convinced am I of the delicacy of this test that I now administer arsenic with a daily examination of urine, except in cases in which the dosage of arsenic is exceedingly small, approximating what is for the sake of a better term called tonic dose.

"The mildest cases of chorea minor in which there are twitchings to a limited degree, and in which the patient is able to be up and about, are best managed by a modified rest cure. Before rising a light breakfast is taken in bed. Before breakfast the patient is given a warm bath and a cold water rub down, is put to bed, and after an interval of an hour partakes of the above light breakfast. The patient is then rested and at 10 a. m. arises and is indulged in the ordinary duties of childhood. At midday, after luncheon, the patients are encouraged to rest whether they sleep or not for an hour, and they may again indulge in mild play or read. Early in the evening the patients are put abed (5 p. m.), and at 6 p. m. a light supper is partaken of, and after an hour of reading or occupation in bed the little ones are encouraged to sleep.

"The moderately severe cases are those in which the chorea is seen to interfere with the mental stability of the patients; they become morose, melancholy, or are subject to outbursts of temper. Such cases are put abed, but not completely isolated; they should be surrounded with quiet, but even here a little play is useful therapeutically. It is unnecessary to darken the room except when asleep or inviting sleep. If there is slight or marked cardiac involvement no arsenic should be given; such cases do very badly under arsenic therapy; the appetite is interfered with, and I really believe the cardiac condition is aggravated by the stomach distress and nausea caused by physiological dosage of the drug, not to mention the kidneys. In these cases, if well borne, moderate doses of sodium salicylate are useful for the alkaline effect only and possibly as a prophylactic if there is a rheumatic tendency. If the salicylate of sodium is badly borne, no drug beyond a sedative given in the mild cases should be used, nor should digitalis be given to drive the heart. Of great use in these cases, it has seemed to me, was the administration of strychnia as a bitter tonic.

"The severest forms of chorea minor are those in which speech is lost. The children are mentally depressed, there is absolute loss of coördinate motion, there is a constant choreic motion of all the body, and in severest forms there is a paresis in which the extremities either cannot be moved at all or but to a limited extent; any attempt to move them only calls forth choreic movements of the face, tongue, and trunk. Some of these cases can neither raise nor lower the extremities. Such cases cannot support the body weight, they are for the nonce paralytic, and are indeed called paralytic chorea. In most of the cases of this class there is no endocarditis, though the heart action may be extremely weak. In these cases I think the use of arsenic is fraught with danger, for, though we have means of judging the limits to which the drug may be pushed, we may go a little too far and add a neuritis to our already helpless picture. In these cases more complete isolation is indicated than in cases of the previous class, no communication should be allowed with other children, and yet there should be sunshine and air. Inasmuch as some of these cases at first or in course of the disease are thrashing about in bed and are apt to injure themselves, the sides of the bed should be padded and the knees and elbows bandaged so as to avoid abrasions, which otherwise would occur. In girls the hair should be neatly done up in a species of cap to avoid matting. Here the sedative treatment, hydrotherapy and strychnia, lend their best aid to the physician."

Chorea Gravis. R. W. Phillip[1] reports a case of chorea gravis, ending fatally. "On examination, the patient presented the aspect of intense motor restlessness. The clinical picture might truly be described as one of muscular insanity *(folie musculaire)*. The motor contortions were universal and most disorderly. The face showed every variety of grimace and contortion. The forehead was repeatedly wrinkled and alternately knit. The eyebrows were similarly raised and depressed, independently of the movement of the forehead, and often independently of each other. The eyelids were closed and opened irregularly, unequally, and often independently of each other. The eyeballs were constantly moved in every possible direction—upwards and downwards, from side to side, obliquely, sometimes in conjugate fashion, sometimes independently. In addition to this, there was present what I do not remember to have seen described anywhere in relation to chorea, namely, the condition of *hippus,* that is to say, the pupil was alternately dilated and contracted in extraordinarily rapid fashion." (See Langmead's article, page 52.)

As to *treatment*, "it is desirable to have the patient on a soft mattress, preferably a water-bed; all hard structures in his neighborhood must be carefully padded; the head and corners of the bed should be cushioned; the patient's elbows and wrists, knees and ankles should be swathed in cotton wadding. It is not a bad plan to attach a pillow to the inner aspect of one of his legs. Scrupulous care must be taken in relation to feeding. The food should be of a simple, nutritious kind. I am much in favor, in such cases, of the systematic exhibition of egg-flip every 2 or 3 hours. Alcohol, perhaps best of all in the form of brandy in egg-flip, say, ℥ij, is of much service. Hydrotherapeutic measures may prove serviceable. Where the movements are not too violent a warm bath is of service. In other cases the wet pack may be used with benefit. In all cases great care should be taken in the matter of

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(1) Brit. Med. Jour., Feb. 15, 1908.

cleanliness, more particularly in relation to scratches or sores of mucous membrane and skin which may be produced through the violence of movement.

Chorea Gravis and Septicemia. B. Sachs[1] discusses the relation between chorea gravis and septicemia and reports 3 cases, 2 of them with postmortem findings. Like epilepsy, chorea has become a generic term, and chorea is a genus with many species, of which infectious chorea is one. Sachs makes the following summary of his cases: "My patients were 15, 18, and 22 years old. After a somewhat indefinite prodromal period of restlessness and slight twitchings, universal choreic movements of an extravagant type set in, which constitute the most prominent symptom of the disease. The jactations are so severe that the patients have to be restrained or have to be carefully watched over in padded beds. Erosions appear all over the body and particularly on the parts most apt to be rubbed or bruised by contact with the bedclothes and the bed posts. The erosions are found chiefly on the buttocks, the elbows and the ankles. Speech is difficult at an early stage of the disease and later becomes impossible. This is due in part to the extreme choreic movements of the lips, of the tongue, and of the muscles engaged in the mechanism of speech, also to the accompanying mental change which may vary from mild stupor and apathy to a condition of active delirium. The movements are lessened during sleep, but sleep is fitful and, as a rule, can be induced only by large doses of hypnotics and sedatives. After a period varying from 1 to 2 weeks, the patient passes from the state of continued restlessness into stupor and coma. After a further period of a few weeks, death supervenes. But I would not claim that a fatal termination is inevitable. High fever is observed from the beginning to the end of the disease. A universal erythema bearing all the marks of a toxic eruption occurs in the earlier stage.

"Blood cultures made in the first case on 3 separate occasions all showed *Staphylococcus aureus*. Dr. Libman, who made the postmortem examination, reports as follows: 'Evidently there was at first a systemic invasion of the

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(1) **Med. Record, March 28, 1908.**

Staphylococcus aureus with an establishment of an acute endocarditis. The port of entry for the infection was at the laceration of the cervix. This invasion was receding when apparently there was a secondary invasion of streptococci from the bedsore. The first infection cannot be attributed to the bedsore, which did not exist at the time when the *Staphylococcus aureus* was first found in the blood. Streptococci were found in large numbers in the spreads at the postmortem examination. The fact that organisms could not be cultivated from the heart blood at the time of the postmortem examination is to be explained on the theory that most of the bacteria lost their vitality during the time that the body lay in the ice-box before the postmortem was made.'

"Referring once more to the choreas as such, the question arises whether there is, after all, any relationship between what we have called septicemic chorea and ordinary chorea minor. The same nerve elements, assuming for the time that they are the motor neurones in the cortex and elsewhere, are affected, in the one case possibly in a purely functional manner as a result of exhaustion, in the other case by the poison acting upon these same neurones. The parts affected may be the same in both diseases, but the disease process would seem to me to be an entirely different one. Nor can one claim that septicemic chorea is merely an intensification of the morbid process giving rise to chorea minor. To my way of thinking a new and independent infection has been, or may be, superimposed, upon a preceding chorea minor. The nerve elements involved in the latter fall victims most easily to the influence of a toxin circulating in the system, and in this way we may account for the fact that in several of the cases reported mild attacks of chorea minor have preceded the grave attack culminating in death. We must not overlook the fact, furthermore, that the constant abrasions of the skin brought about by the frequent movements of the choreic patient yield easy and innumerable points of access for the invading organisms.

"As far as the *treatment* is concerned, it has been unavailing to the present time. Salicylates, sedatives, hypnotics, and the usual forms of hydriatric treatment have been employed. The good results that have been obtained in other severely toxic and infectious disorders by the use of appropriate antitoxins, leaves us some hope for the future. Meanwhile we must endeavor to eliminate, if possible, toxic products circulating in the body; the wisdom of such a procedure was shown in the recently reported case of Ewald and Witte."

George Carpenter[1] reports a fatal case of chorea gravis associated with *double optic neuritis* and hyperpyrexia (106.4° F.) in a child aged 3½ years. Carpenter states that this case presents the only rheumatic hyperpyrexia in a child that he has ever seen in a rich experience extending over 20 years. The postmortem, however, showed only a slight thickening of the mitral valve, and a normal brain.

DISEASES OF THE BRAIN AND MENINGES.

Rheumatic Cerebral Meningitis.

Rheumatism of the Brain. Rheumatic cerebral meningitis. Lépine[2] reports 2 cases of what he calls cerebral rheumatism, though it is very doubtful whether such a morbid entity, described even by Lépine, will be generally credited. He begins by saying that in 1864, before the salicylic acid treatment of rheumatism was introduced, at a time when huge doses of quinin (2 to 4 grams per diem) were given, cerebral rheumatism was common. The symptoms were, however, attributed to the quinin rather than to the brain. After 1876, when the salicylates were introduced, cerebral rheumatism dropped out of notice, so that in the last 30 years Lépine has seen but 2 cases. The first of these he saw in 1878, and a brief summary of this case is reported. The second case, a recent one, occurred in an Italian, 30 years of age. He was suffering from acute articular rheumatism, and entered with a temperature of 40° C. (104° F.). Though he had lived in France since he was 10 years old, he replied badly to questions. The joints affected were only moderately

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(1) The Lancet, Nov. 30, 1907.
(2) Lyon Méd., July 12, 1908.

swollen, and were not very painful. The heart gave no evidence of endocarditis. Two days later the temperature had fallen to 37.5° C. (99.5° F.), but he still seemed unclear mentally. On the next day a slight delirium developed, and the externe, thinking it due to the salicylates, discontinued them. His temperature again rose rapidly to 104° F. without any increase of joint complications, and the delirium became stormy. The next evening at 6 he died with a temperature of 42.5° C. (108.5° F.). At the autopsy the meninges were found reddened, but were easily separated from the cortex. Over a certain number of the gyri there was a reddish coloration, which did not disappear when washed by a stream of water. The brain was congested. There was no appreciable increase of the cerebrospinal fluid, and the ventricles were not dilated. These findings Lépine interprets as indicating rheumatism of the brain, which occurs, he says, in untreated or insufficiently treated, cases.

CARCINOMATOSIS OF THE MENINGES.

D. J. McCarthy and M. K. Meyers report a case of carcinomatosis of the meninges, with a consideration of the diagnosis of multiple carcinomatosis, tuberculosis of the nervous system, disseminated syphilis, and multiple sclerosis.[1] The condition is very rare, only 11 cases being discovered by the author after a careful search of the literature.

The case here reported occurred in a colored cook, whose illness began 3 months previously with pain in the right elbow and hand, and headaches in the occipital and temporal regions. Two days later the pain extended to the left hand. On examination, normal motion was found in all 4 extremities, and sensation was everywhere perfect. The only cranial nerve disturbance was a sluggish reaction of the right pupil to light. The right knee-jerk was absent, the left diminished. There was no ankle-clonus and no Babinski reflex. The superficial abdominal reflexes were normal. The eye grounds were normal and the nerve-heads of good color. A month later the left

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(1) New York Med. Jour., Feb. 1, 1908.

sixth nerve became paralyzed, and the muscles of the left leg were found to be more flabby than those of the right. A well defined Babinski reflex had also developed. Five days later the patient became semiconscious, with double partial ptosis and choking spells on swallowing. Two days afterwards she died.

The postmortem examination showed "chronic pleurisy, hemorrhagic infection of the lung, carcinoma of the lung (primary), chronic diffuse nephritis, secondary carcinoma of the liver, and primary carcinoma of the pancreas." * * * "The entire brain was covered with minute white areas, varying in size from a pin point to a pin head. These areas were most frequent along the course of the bloodvessels, and occurred in scattered points over the entire meninges. They were most marked and occurred in the greatest numbers over the base of the brain, and especially in the interpeduncular spaces."

Plate I. beautifully illustrates this condition.

"Microscopical examination: Cerebral cortex. The tubercles of the cerebral meninges were composed of a reticulum of connective tissue containing large masses of a columnar type of epithelial cells, arranged in cell nests, and having the appearance of a carcinoma." There was no degeneration of the spinal cord by the sheath stains, but other methods showed infiltration of the meninges and marked secondary degeneration in the posterior columns of root origin. There was also some degeneration of the direct pyramidal tract of the left side, and a few scattered dots here and there in the right crossed pyramidal tract.

"A diagnosis of cerebrospinal syphilis was made in this case, in spite of the fact that the patient presented no history of syphilis. This negative symptom, if it can be so called, was not given its proper value. This is not a matter of surprise when there is taken into consideration the difficulty in securing a positive history of syphilis, even when the patient is aware of it." A diagnosis of disseminated tuberculosis, in view of the lung findings (not here reviewed), would have been a better diagnosis. Or even multiple sclerosis might well have been considered.

LEAD MENINGITIS.

M. Pinard[1] again calls attention to the fact that lead encephalopathy is only another name for lead meningitis, as has been proved by lumbar puncture by Mosny and Malloizel.[2] Lumbar puncture has revealed the extreme frequency of meningitis reactions to lead, and has permitted the separation of a distinct group of cases of lead meningoencephalitis from other lead cerebral syndromes depending on various changes, most usually vascular. In the lead meningoencephalitic group lumbar puncture constantly shows a lymphocytosis of the cerebrospinal fluid. Some of these cases are very mild, while others are accompanied by a stormy symptomatology.

Latent lead meningitis without appreciable clinical symptoms is the most frequent, and can be demonstrated by lumbar puncture in almost all cases of lead colic. It is purely a histologic meningitis, without a nervous symptom. Lumbar puncture shows a slight hypertension and an almost pure lymphocytosis:—8 or 10 lymphocytes being found to a field after centrifuging. Sometimes as many as 30 to 50 cells per field may be counted. At times this latent form may show a few abortive symptoms, chief of which is headache, which may be accompanied by insomnia, restlessness, and dullness. After a few days the headache may disappear. Even vomiting has accompanied these headaches, and also exaggeration of the tendon-reflexes. When these graver symptoms appear the case is really a *forme fruste* or abortive form of cerebrospinal meningitis.

Acute or subacute attacks of lead meningitis may be late or early. The early form usually appears in workmen exposed to rapid and intense intoxication, the symptoms depending upon the location of the preponderating lesion, whether at the base, on the convexity, or along the cerebrospinal axis. Among some, bulbar signs predominate; others show epileptiform seizures, either isolated or in the course of a more or less complete meningitic syndrome; still others show mental phenomena of excitation; or signs of spinal irritation may dominate the

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(1) Gaz. des Hôp., July 18, 1908.
(2) Revue de Méd., June, 1907.

scene. Thus there is the complete meningitic form, the epileptic form, the psychic form, and the spinal form among these early acute attacks. The symptomatology of each of these forms is carefully set forth by the author.

The complete *meningitic form* resembles tubercular meningitis. There is headache, vomiting, and constipation, with a low fever, a slowed pulse (50 to 60 beats per minute), contractions of the legs and arms, sometimes absence though usually the presence of Kernig's sign, delirium, incoherence, even convulsions. Although rare, strabismus, amblyopia, loss of the pupillary reflex to light, and inequality of the pupils, may all occur. The blood shows marked anemia and basophilic granules in the erythrocytes.

The *epileptic form* is by no means infrequent. Convulsions may occur alone, or only with headache, or with numerous meningitic symptoms. They are usually generalized, but may be so slight as almost to be masked. Usually the convulsion follows an attack of lead colic and is attributed to it.

The *psychic form* is distinguished by mental states reminding one of toxic mental aberrations; there is delirium, loss of the reasoning faculty, and the patient talks constantly with imaginary persons. After a period of excitement comes one of depression, and then the mental equilibrium is regained. The *spinal form* is devoid of headache or any cerebral symptoms whatever. There are lumbar pains radiating down into the thighs, paralyses, pareses, signs of irritation of the pyramidal tracts, exaggerated knee-jerks, ankle-clonus, sensory disturbances, such as anesthesia, analgesia, and thermanesthesia. In general the disturbances are not severe, and may seem inconsequential unless thoroughly investigated.

The *late* variety of acute or subacute lead meningitis exists in two forms. (1) The first is known as general lead paralysis, and is a diffuse meningoencephalitis comparable to syphilitic general paralysis. It is, however, susceptible of regression, but often steadily progresses as in a case reported by Sicard. (2) The second form can be called general lead pseudoparalysis, and is not only regressive, but susceptible of complete cure. Among the prodromata are headache for a month or two, followed by depression, preoccupation, taciturnity, emotionalism, conversation with imaginary people, especially in the night. There is also a general muscular numbness, fibrillary twitching, and tremor. At times there is complete lucidity, perfect memory; at other times, obsessions and mental confusion. There is never dementia. Speech is disturbed, reading aloud is exhausting, writing is tremulous, and letters are omitted. The reflexes are exaggerated and hyperesthesia is the rule. Each of these forms may become more or less chronic, and become indistinguishable ultimately from ordinary cases of general paralysis of the insane. Very important is it to inquire into the matter of alcoholism and syphilis, for these are frequently associated with lead poisoning and complicate the pathology. The diagnosis can be based *only* upon the results of lumbar puncture.

TYPHOID CEREBROSPINAL MENINGITIS.

A case of purulent cerebrospinal meningitis caused by the typhoid bacillus, without the usual intestinal lesions of typhoid fever, is reported by J. Norman Henry and R. C. Rosenberger.[1] The patient, a colored man, 34 years of age, complained of headache, dizziness, constipation, and fever, with vomiting. He became delirious on the ninth day of his illness. Severe pain in the back and legs was his complaint, and there was nose-bleed. His head was slightly retracted, and he was very restless. There was at the time he became unconscious a slight ptosis of both lids, and the eyes were drawn upward and to the left. The pupils were small, equal, and did not react to light. The pulse was rapid, regular, and of high tension. Lungs and heart were normal. The spleen could not be felt. The spine was rigid and slightly concave. Kernig's sign was present. Lumbar puncture showed a turbid fluid with a heavy, purulent sediment, examination of which showed a bacillus which looked like that of typhoid fever. The Widal reaction was suggestive, but the leucocyte count showed 14,000. A count of the fluid showed 96 per cent. of polymorphonuclear cells and 2 per cent. each of lymphocytes and hyaline cells.

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(1) Am. Jour. of the Med. Sciences, February, 1908.

Careful study was made of the fluid by inoculations into various culture media, and a bacillus was discovered which closely resembled the typhoid bacillus in every characteristic. It was agglutinated in dilutions of 1 to 40 with a known typhoid serum, and with the serum of a typhoid patient. Five c.c. of the blood gave a positive culture in 200 c.c. of bouillon. This culture was also agglutinated in dilutions of 1 to 40 with the patient's serum, and also with a known typhoid serum.

At the autopsy, spreads and inoculations were made from the pus upon the brain, and these were found to contain an organism identical with that isolated from the blood and from the cerebrospinal fluid. The postmortem examination showed the spleen to weigh 110 grams, possessing a thin and smooth capsule, and normal appearance in section. The small intestines were normal throughout. The large intestine was found to be also normal, although the mucous surface was moderately congested in its upper part, but with no trace of ulceration. Peyer's patches were enlarged. The dura mater was found tense and congested; upon removal, the pial vessels were found deeply injected, and outlined by yellowish lines of pus. There was no excess of meningeal fluid. At the base of the brain, about the pons and medulla, extending into the spinal cord, a large amount of pus welled up, as from an abscess. No pus was found in the middle ears. The dura over the base showed no evidence of extension of disease from the nose. The cord was covered throughout, especially in its upper two-thirds, by a purulent exudate like that found at the base and on the convexity of the brain.

The conclusion that this is a case of purulent cerebrospinal meningitis caused by the typhoid bacillus seems unavoidable and incontrovertible. Very unusual indeed is the absence of the customary intestinal lesions of typhoid fever. In Cole's review of the cases of typhoid meningitis in the literature,[1] fourteen in number, the exudate was variously purulent, fibropurulent or hemorrhagic and purulent. In all, however, general typhoid lesions were found present, except in one case where no autopsy was

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(1) Johns Hopkins Hospital Reports, 1905.

made. A case like this one now reported, without the usual typhoid lesions, was reported by Neumann and Schaeffer, though these authors hesitated to affirm that the organism isolated by them, though greatly resembling the typhoid bacillus, was positively the Eberth bacillus.

EPIDEMIC CEREBROSPINAL MENINGITIS.

Etiology and Symptomatology. The etiology and symptomatology of cerebrospinal meningitis are presented by T. A. Clayton.[1] The disease is met with from Montreal to Mobile in the western hemisphere, and from Sweden and Russia to Algiers and Jerusalem in the eastern.[2] It is probable that overcrowding and bad hygiene, necessitated among the poorer classes in cold weather, accentuates its appearance at that season. Neither sex, race nor social condition seems to be of importance. One attack does not confer absolute immunity, since there are instances in which individuals have suffered from two attacks. Among predisposing factors, age is the most important, children and young adults furnishing the large majority of victims. Taking the figures given by Hirsch, Westenhoeffer, French and Taves, it is found that of 4,584 cases, over 78 per cent. were 15 years of age or younger.

Important as is the matter of its communicability, we are in almost total ignorance concerning it. It has never been proved that the disease is contagious in the ordinary sense. Not a few observers have likened it to pneumonia in its communicability. Numerous instances are on record in which two or more persons have been stricken in the same house. Neither are we aware of the mode of entrance of the organism, and its pathway to the brain and cord. Most plausible is the theory of entrance through the nasal mucosæ, and possibly the cribriform plate of the ethmoid.

Symptomatology. The severe type sets in suddenly, usually in the midst of health. Intense headache is the first symptom. The pain may be general or occipital, constant or remittent, with exacerbations. Pain may ex-

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(1) Am. Jour. of the Med. Sciences, February, 1908.

(2) An epidemic occurring in the Soudan was reported in the Practical Medicine Series for 1907, p. 50.

tend down the back and around the body into the abdomen or joints. Vomiting is likely to occur. In the fulminating cases, delirium, unconsciousness, and coma, with or without convulsions, rapidly ensue. Convulsions are much more common in children, and the younger the child, the more likely the convulsions. Rigidity of the post-cervical muscles, with retraction of the head, is almost always observed. The patient ordinarily lies on the side, with the head thrown far back, thighs flexed on abdomen, and legs on the thighs. Paralyses are often present, such as ptosis, strabismus, monoplegia, and occasionally a complete hemiplegia. The tendon reflexes are variable. Kernig's sign is of great importance, but is not pathognomonie. Hyperesthesia is usual, especially in the earlier stages. Irritability and restlessness are the rule. Photophobia is often observed. Incontinence of urine and feces is frequent, or there may be complete retention. Constipation is almost a constant symptom. The fever and pulse are variable. Herpes and petechiæ occur in about 25 per cent. of all cases. Urticaria, erythema, rose-spots like those of typhoid fever, bullous, vesicular, and measly eruptions, have been met with. A leucocytosis is always present, with a relative increase of polymorphonuclear leucocytes. The cerebrospinal fluid is usually turbid or purulent, but may be clear.

Diagnosis. In the Practical Medicine Series, 1907, Vol. X, p. 49, attention was called to cases of cerebrospinal meningitis in which the *Diplococcus intracellularis* was found in the blood. J. M. Birnie and M. T. Smith[1] first call attention to Simon's article, and then report a case of their own in which the diagnosis was made from a blood-culture. The patient, a girl 15 years of age, with symptoms suggestive of the disease, showed a diplococcus in a blood-culture made from 4 c.c. of blood. The diplococcus proved to be a Gram-negative one, growing on agar. A lumbar puncture was then performed, and the same diplococcus found in the cells of the cerebrospinal fluid. The patient was treated by the injection of 0.5 c.c. of a "personal vaccine" standardized at 20,000,000 to the cubic centimeter. Improvement followed, and ultimately com-

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(1) Am. Jour. of the Med. Sciences, October, 1907.

plete recovery. No statement was made as to whether more than one "vaccine" treatment was given.

H. Conradi[1] suggests that the best culture-medium for meningococci is cerebrospinal fluid. He collects from 10 to 20 c.c., centrifuges it, and separates it from the sediment. He then heats it to 60° C. for one to two hours. To one part he adds three parts of agar cooled down to 40°, and this he pours into petri-dishes. On this he makes his cultures out of the coagulum after centrifuging. A moist chamber is made, and in this the meningococci grow as well as upon ascites-agar.

Westenhoeffer[2] discovered in his experience in epidemic cerebrospinal meningitis in Silesia, that cultures of meningococci could frequently be obtained from the pharyngeal tonsil rather than from the nasal secretions. The involvement of the throat and pharynx is very rapid, being in its most acute stage and most evident in the first week of the disease. Examination shows the meningococci to be far back in the upper part of the pharynx, and when looked for here they will be found. Formerly only the nasal secretions were examined, and hence the meningococci were frequently overlooked. If, however, every case of apparent epidemic cerebrospinal meningitis is examined as to the bacteria in the pharynx, the meningococci will be found, and can be cultivated. Thus examination of the pharynx bacteriologically must be made when cerebrospinal meningitis is suspected, since it may give positive results before they are given by the cerebrospinal fluid.

The *differential diagnosis* between epidemic cerebrospinal meningitis and other forms of cerebrospinal cases is discussed by Hölker,[3] of the Charité Hospital of Berlin. Since January, 1906, the second medical clinic at the Charité has had 15 cases entering with the provisional diagnosis of cerebrospinal meningitis. Of these, in three the diplococcus meningitidis was found on the first day, and in a fourth tubercle bacilli were found. In two more the meningococci were found within two days' time. There remain 9 other cases, which illustrate the difficulty of diagnosis. Of these 9, 2 showed meningococci in their

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(1) Deutsche med. Woch., July 9, 1908.
(2) Berl. klin. Woch., Sept. 23, 1907.
(3) Berl. klin. Woch., Aug. 26, 1907.

cerebrospinal fluid rather late—one at the end of the second and one at the end of the fifth week. The latter case, constantly suspected as epidemic cerebrospinal meningitis, occasioned some doubt in diagnosis by the very reason of the continued absence of the meningococci.

Another case, developing suddenly in a perfectly healthy individual 30 years of age, beginning with headache, vomiting and retraction of the head, a marked Kernig's sign being present, the cerebrospinal fluid showing leucocytes and lymphocytes and diplococci, part of which were intracellular, capsulated and Gram-negative, turned out to be *meningitis tuberculosa purulenta* at the postmortem examination which was held.

In the remaining six cases examination of the cerebrospinal fluid for bacteria constantly gave negative results. In one of these syphilis was ultimately regarded as the cause of the meningeal irritation. The other five, all resulting fatally, were examined postmortem. One was a tumor of the brain (sarcoma), one a hemorrhagic diathesis with cerebral hemorrhages, one was a purulent meningitis, and two were cases of tubercular meningitis. Of these latter, one case had lumbar puncture performed on him 7 times, and the cerebrospinal fluid showed numerous leucocytes and only a few lymphocytes.

Therefore in 15 apparent cases of cerebrospinal meningitis epidemica on the day of entrance, examination of the cerebrospinal fluid showed promptly, or after some delay, one case to be tubercular cerebrospinal meningitis and 7 to be the epidemic variety. Of the remaining 7, the diagnosis was not once settled by examination of the cerebrospinal fluid. Certainly the rule that leucocytes means the epidemic variety and lymphocytes the tubercular or syphilitic variety is subject to numerous exceptions in both ways. Very significant in these cases is the fact that the meningococcus may not be found till late in the disease.

Serum Therapy. (a) *Wassermann Serum.* A. Wassermann[1] defends his serum for the cure of epidemic cerebrospinal meningitis. He has furnished it liberally to those asking for it, and, out of 1,037 samples given away, received 102 replies as to patients treated. Of these, many

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(1) Deut. med. Woch., Sept. 26, 1908.

are imperfectly reported or no microscopic examination was made, so that only 57 can be used. Of these 57 treated with serum, 27 died, or a mortality of 47.3 per cent.

If these cases are computed on the basis of the date the serum was given, the following results are obtained: Mortality in 14 patients given serum on the first or second day, 21 per cent.; mortality in 7 patients given serum on the third day, 28.5 per cent.; mortality in 7 patients given serum on the fifth day, 28.5 per cent.; mortality in 4 patients given serum on the sixth and seventh days, 75 per cent. The later the serum is given, the higher the mortality, reaching 80 per cent. when given on the fourteenth and fifteenth days. The mortality of cases treated by the serum during the first week is 35.1 per cent., as against 55 per cent. so treated during the second week. Wassermann believes these figures justify the continuation of his serum therapy, and urges that the serum be given early. The amount which may be given is large, 10 c.c. being given to 2-year-old children. One adult received in 7 injections 130 c.c., given in seven days. In no instance have bad effects from the serum been reported. The serum may be given subcutaneously or be injected directly into the spinal canal, first taking care to extract cerebrospinal fluid a little in excess of the amount of serum to be introduced, to avoid evil pressure-consequences. The effect of the serum is not so much a reduction of the temperature as an improvement of the general condition, the relaxation of the rigidity of the neck, the passing away of the headache, stupor, restlessness, and cerebral symptoms. When relapses occur the serum should be freely used, as in the beginning.

The author's conclusions follow: The serum is to be used in rather large amounts, frequently repeated, given subcutaneously or injected into the intradural space. It is harmless, even for children. The serum, if it is to be successful, must be given early. When the disease is subacute or chronic, the serum is not likely to be of advantage. The injections must sometimes be repeated daily, according to the course of the case. In very severe cases it is recommended that the serum be injected directly into the spinal intradural space. Before making such an injection, cerebrospinal fluid in amount in excess of the serum to be introduced should be withdrawn. The single dose in children from 1 to 2 years of age should never be less than 5 c.c., and in older children and adults never less than 10 c.c. These doses may be repeated twice or thrice even on the same day. The use of the serum should be continued for several days even where improvement is noted, since the improvement after a single injection is only transitory, and the disease is prone to relapses.

Werner Schultz[1] during the period from April, 1906, to April, 1907, treated altogether 64 cases of cerebrospinal meningitis in the City Hospital of Posen. Of these, 23 were treated by the Kolle-Wassermann serum, and the results obtained are compared with the results obtained by the method of treatment of the 41 other cases. The injections of serum were made subcutaneously at the earliest possible moment, in doses of 10 c.c. in adults and 5 c.c. in children. The dose was repeated if necessary, even up to four times. The results as to control of the fever were poor. Of the 23 cases treated with the serum, the fever was uninfluenced in 16, and raised or lowered equally in the remaining 7. As to mortality, 56.5 per cent. of those treated with serum died, as compared with a mortality of 53.7 per cent. in those otherwise treated. Early injection did not favorably influence the prognosis, 53.3 per cent. of those given serum during the first week of illness resulting fatally. Schultz concludes that the Kolle-Wassermann serum is of no value in the treatment of epidemic cerebrospinal meningitis.

(b) *Flexner Serum.* A. Miller and S. A. Barber,[2] of Porterville, Cal., report an epidemic of cerebrospinal meningitis, consisting of 16 cases. Of these, the first 12 were not treated by Flexner's antiserum, and only one of the 12 recovered. The remaining 4 were treated by the antiserum, and only one of these died. The whole 16 histories are given, the last 4 in detail. The authors state that their small experience teaches that the serum should be used as early as possible, and that it should be injected daily into the spinal canal in doses of 30 c.c., whether the same amount of spinal fluid is removed or not.

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(1) Berl. klin. Woch., Dec. 30, 1907.
(2) Jour. Am. Med. Assoc., June 13, 1908.

F. S. Churchill[1] reports 11 cases of cerebrospinal meningitis treated by Flexner's antimeningitic serum; of these, 7 recovered and 4 died. Of these 4 cases, only 2 were proved by bacteriologic examination to be of the meningococcic type. Thus in 7 of the 9 proved cases the patients have recovered.

"The technic of giving the serum is simple. A lumbar puncture is done and, if possible, 30 c.c. of fluid is withdrawn. Then to the cannula still in place is attached an ordinary antitoxin syringe, the serum poured from the bottle into the barrel of the syringe and allowed to run directly into the spinal canal. As a rule, the piston must be used to force at least the last part of the serum. The serum should be heated to body temperature before using. This may be done by standing it in warm water. Flexner recommends, for the present, a dose of 30 c.c., to be repeated every 24 or 48 hours, for 3 or 4 times if there be no improvement. It also seems safer to withdraw spinal fluid equal in amount to the serum to be injected, to avoid pressure symptoms. Some cases have been reported, however, in which no fluid whatever has been withdrawn and as high as 20 c.c. of the serum injected without bad results. If for any reason it is found impossible to inject into the spinal canal, the serum may be given subcutaneously, but good results are not expected by this method."

Churchill's conclusions as to the effect of the serum upon the general condition and the temperature are favorable. The first sign of improvement in at least 5 of the patients was a clearing up of the mental condition.

"The effect of the serum on the total leucocyte count was striking and uniform. There was almost invariably a drop after the first injection, sometimes to a marked degree—e. g., in Case 6, from 19,000 to 13,000 within 24 hours after the first injection; Case 5, from 22,000 to 17,000; Case 10, from 23,000 to 14,000. In Case 11, a very severe one, the first injection failed to produce any effect on the general condition, and the disease apparently continued to advance, for we see in the first 24 hours a jump in the leucocyte count from 18,000 to 29,000. Then the second injection evidently began to produce its

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(1) Jour. Am. Med. Assoc., July 4, 1908.

effect, as was seen by the brighter mental condition of the patient and a considerable drop in the leucocyte count, from 29,000 to 15,000.

"The number of cases reported in this paper is, of course, small, and taken by themselves would be of no significance. Taken with the cases already reported by Flexner and his other investigators, the results of this treatment of meningococcic meningitis are most remarkable. As one watches the behavior of these patients after one, two or three injections of this serum, he is impressed with the astonishing change which comes over them, the clearing mentality, the unsuffering expression of countenance, the evident comfort, even with a still retracted head and rigid muscles, and he is filled with an optimism as to the ultimate result which perhaps is not yet justified."

C. H. Dunn[1] discusses the serum treatment of epidemic cerebrospinal meningitis based on a series of 40 consecutive cases, all treated by Flexner's antimeningitis serum. All cases in which the *Diplococcus intracellularis* was found in the cerebrospinal fluid, without regard to the type of case or stage of disease, are included in the series. The antiserum was administered in all cases by injection into the cerebrospinal canal according to the technic recommended by Dr. Flexner. As soon as a suspected case was reported, lumbar puncture was at once performed. If the cerebrospinal fluid obtained was cloudy, the antiserum was injected at once, without waiting for the bacteriologic examination; and if the fluid was clear, no antiserum was given until subsequent examination revealed the *Diplococcus intracellularis.* In this way some cases of pneumococcic and streptococcic meningitis were given the antiserum; but without any harm being done to the patients. These cases are not included in the series, but are mentioned merely to emphasize the fact that the serum is harmless and that the advantages to be gained by the earliest possible use of the antiserum in epidemic cerebrospinal meningitis are great enough to outweigh any possible disadvantage from its use in other forms.

"In those cases in which a rapid and marked improvement in the symptoms occurred after the first injection,

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(1) **Jour. Am. Med. Assoc., July 4, 1908.**

accompanied by a permanent fall of temperature to the normal, no further injection was given. In those cases in which this did not occur, the injections were repeated daily until nervous and subjective symptoms were completely relieved and the temperature had reached the normal, or until four doses had been given. In resistant or relapsing cases, further doses were given according to circumstances. The routine dose was 30 c.c.; in many instances larger doses were given, the maximum being 45 c.c. In some instances, when the amount of fluid obtained was small, and in all instances when too great an increase of intradural pressure was feared, smaller amounts were injected, the minimal being 10 c.c. This was the general routine; there were some exceptions.

"The serum has been used in forty-five cases of epidemic cerebrospinal meningitis, but cases still pending are not included in the series. Of the forty finished cases, nine patients have died and thirty-one have recovered. This is a mortality of 22.5 per cent, and a rate of recovery of 77.5 per cent. Of the thirty-one cases in which the patients recovered, two were left with sequelæ, one being deaf and one both blind and deaf. The recovery was complete in twenty-nine cases, or in 72.5 per cent.

That the forty cases did not all belong to the mild type is shown by the following analysis of the cases:

Type.	Cases.	Deaths.
1. Fulminating	3	1
2. Severe	6	2
3. Common	19	1
4. Mild	6	0
5. Late chronic	6	5

Limitations of the Serum. "The nine fatal cases in the series throw a certain amount of light on the limitations of the value of the serum. Of these nine, five were cases seen late in the course of the disease, at a time when the patients were in the well-known chronic stage, unconscious, and without fever or active symptoms. In one of these the serum was not given until the patient was actually moribund. Of the other four cases, one was of the fulminating type, one a very severe case, and one patient died of an intercurrent bronchopneumonia, coming on after the temperature had come down to normal and all meningeal symptoms had subsided. The last case was one of average severity, in which the serum appeared to produce a slight improvement in the beginning, but which later appeared uninfluenced by the antiserum, advanced into the chronic stage, and a fatal result ensued after many weeks of illness.

"*Effects of the Serum.* The three principal effects of the serum seem to be: First, to produce a fall of temperature; second, to produce a rapid improvement in the patient's general condition, accompanied by a more or less marked relief of certain symptoms, and, third, to cut short the course of the disease.

"The most striking effect on the temperature is a permanent critical fall following the first dose of serum. This occurred in ten cases, the temperature falling at once to normal, or nearly normal, and showing no further tendency to rise. Other cases showed a similar critical fall of temperature, which was, however, not permanent, the temperature rising again, and finally coming down by lysis. Such a temporary crisis occurred in four cases. Another effect was seen in a rapid permanent lysis, which was very striking in cases in which there had been a consistent high temperature up to the time when the first dose of serum was given. This occurred in nine cases. A similar lysis, but not permanent, the temperature going up again, occurred in two cases. A temporary sharp fall of temperature, not to the normal and not permanent, occurred in four cases; in these the temperature rose again, to come down by lysis later in the course of the disease. There was no immediate effect on the temperature noted in 5 cases. In the remaining 6 cases the patient had a normal temperature when the serum was given.

"The effect on the symptoms and general condition is the most striking phenomenon observed with the use of the serum. In severe cases there occurred a permanent return to consciousness, a disappearance of mental dulness, a disappearance of delirium, and a disappearance of headache, hyperesthesia, tenderness of the neck, or vomiting. These symptoms were often relieved completely within twenty-four hours after the first injection, the patient changing in the most remarkable way from a serious condition of coma to a favorable condition of normal mental activity." In the late chronic cases, of which there were 5, no improvement at all took place, and all resulted fatally.

The serum seems to cut the disease short, the average length of time which patients remained under treatment being but a small fraction of the time in which patients recover under the old expectant treatment, when they recover at all. Another striking effect of the serum is exerted in the cerebrospinal fluid. When large numbers of diplococci are found at the time of the first injection, but few will be found when the next inspection is given, and these will be almost exclusively intracellular. The third puncture usually shows still fewer diplococci, and these exclusively intracellular. In the fourth fluid, after three doses, there are frequently no diplococci to be found. In 7 cases there were relapses, five of which responded to one dose of the serum, and two of which responded to three or four doses.

"*Lack of Sequelæ.* The completeness of recovery is another noteworthy point in this series. There were sequelæ in two cases only, one child being deaf and one blind and deaf. The earlier it was employed, the more marked were its effects. In 5 cases the disease was suddenly aborted after one dose of serum. In 4 of these cases the serum was given in the first forty-eight hours. The fifth case was an exception, the disease having run a steady, unchanged course for twenty-one days, with constant fever and symptoms. A crisis took place and there was complete relief of symptoms after one dose of serum.

"The relation between the mortality and the stage of the disease is shown by the following table:

Serum given in first week, 8 per cent.
Serum given in second week, 0 per cent.
Serum given after second week, 77 per cent.

"We may conclude that the prospects of aborting or rapidly cutting short epidemic cerebrospinal meningitis are better the earlier in the disease the serum is given, and that the antiserum usually has no effect in the late chronic stage. There were cases which proved exceptions to this rule, as in three cases in which the serum was used comparatively early the patients died. On the other hand, one patient in the late chronic stage began to improve immediately after one dose of serum and made a rapid convalescence. This would show that there is always some hope of a good result so long as diplococci are present.

"As to the amount of serum which should be given, I believe that 30 c.c. can be given, usually, with perfect safety. One can judge to a certain extent how far it is safe to go by the feeling of resistance to the injection of the serum. In cases in which larger amounts of fluid are withdrawn, I believe 45 c.c. should be given at a dose.

"The daily injection of the serum in most cases seemed to be effective. After four doses have been given, if after one or two days the case proves resistant, or at any time if there is a tendency to relapse, this treatment should be repeated. It is a question whether in some severe cases the serum should not be administered oftener than once in twenty-four hours."

The author concludes as follows:

"1. The use of the Flexner antiserum is of great value in epidemic cerebrospinal meningitis. I believe its value to be comparable to that of diphtheria antitoxin in diphtheria.

"2. The use of the serum at times aborts the disease, frequently rapidly relieves its symptoms, shortens its course, lessens the liability to sequelæ, and greatly reduces its mortality.

"3. The serum should be used as early as possible in all cases, even of suspected epidemic meningitis.

"4. It should be frequently repeated as long as there are symptoms or any tendency to relapse.

"5. Late chronic cases are unfavorable for the use of the serum, but any case in which the diplococci are present has some hope of relief by its use.

"6. Some cases are resistant."

Simon Flexner and J. W. Jobling publish an analysis of 393 cases of epidemic meningitis treated with the antimeningitis serum.(1) The analysis is based only on cases proved to be cerebrospinal meningitis of the epidemic sort by bacteriologic examination. The total number of recoveries was 295, and the total number of deaths 98. Hence there were 75 per cent. of recoveries and 25 per cent. of deaths. Tabulated according to the ages of the patients the following is the result obtained:

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(1) Jour. Am. Med. Assoc., July 25, 1908.

Patients.	Total number.	Recovered.	Died.	Per cent. of deaths.
Under 1 year	22	11	11	50
Between 1 and 2 years	19	11	8	42.1
Between 2 and 5 years	68	52	16	23.5
Between 5 and 10 years	79	70	9	11.4
Between 10 and 20 years	105	80	25	23.8
Over 20 years	87	64	23	26.4
Age not given	13	7	6	46.1

The youngest child which recovered was 1 month old. The latest case of the disease, in a child under 1 year of age, which was treated was in its fourth month when the injections were begun; the child died. The highest mortality was among patients over 20 years of age.

"*Results According to the Period of Injection.* We have also analyzed the histories according to the earliest period of the disease at which the injection of the serum was begun. The histories of 328 cases were sufficiently explicit to enable us to approximate the periods in which the first serum injection was made. We have arbitrarily chosen the three periods that follow in which to group the cases:

Period of injection of serum.	Number of patients.	Recovered.	Died.	Per cent. of deaths.
First to third day	121	103	18	14.9
Fourth to seventh day	100	78	22	22
Later than seventh day	107	68	39	36.4

"In spite of the uncertainties surrounding the period of onset of the symptoms which affect the accuracy of the calculation of the period, the beneficial influence of early injection is rendered sufficiently obvious by the table."

"*Manner of Termination of the Symptoms.* The histories have been sufficiently explicit in 270 instances to enable us to determine the manner in which the disease terminated, that is, whether by lysis or crisis. Of the 270 cases described in the histories, 201 terminated by lysis and 69 by crisis. Hence we have assumed that from 25 to 30 per cent. of the cases treated with the serum terminated abruptly in the manner to which we previously applied the term "crisis."

"Closely connected with the question of the manner of termination of the symptoms, is the question of the duration of the active symptoms of the disease in serum-treated as compared with nonserum-treated cases. Without entering into a discussion of this point, we wish to state that we have analyzed 220 histories of recovered patients with reference to the duration of the active symptoms and found the period to be about eleven days.

"*Influence on Diplococci, Spinal Exudate, and Leucocytosis.* In our first publication on the serum treatment of epidemic meningitis we drew attention to a fact which impressed us as remarkable and significant—namely, that soon after the serum injections were begun the diplococci tended to be greatly reduced in numbers, to disappear from the fluid part of the exudate, to become wholly intracellular (unless they were now entirely absent), to present certain changes in appearance, as swelling and fragmentation, and to stain diffusely and indistinctly, and coincidentally to lose viability in cultures. The later and far wider experience has tended to confirm the views we first expressed based on the effects observed; and, while exceptions occur in which the diplococci disappear or become engulfed and change in morphology or lose viability more slowly, yet the general fact seems securely established. There seems little doubt that part of the beneficial effect of the serum injections must arise from the restriction of multiplication and from the greater phagocytosis of the diplococci.

"Attention was previously directed to the rapidity with which the exudate in the meninges loses turbidity under the influence of the serum injections. This fact has been noted again and again in the subsequent cases in which the treatment was carried out. A fair number of patients in whom the notes state the spinal exudate to have been purulent have recovered, and the rapid clearing of the exudate was observed even in them.

"Closely connected with the rapidity with which the cerebrospinal exudate loses pus cells and returns to a limpid condition, is the state of the general leucocytes of the blood. If the inflammatory emigration into the meninges is arrested, then the number of circulating leucocytes should tend rapidly to return to the normal. The facts at hand, based on many counts of the circulating leucocytes before the injections of serum were begun, and afterward at regular intervals, show a fall, often very rapid, and even critical, in the number of leucocytes in the general blood stream, with which the disappearance of the diplococci and the clearing of the spinal exudate are correlated.

"There remains one more topic to be mentioned. The indications given by the first series of serum-treated cases were to the effect that in the great majority of instances recovery from the disease would be complete. The facts brought out by the far larger series of cases on which this article is based confirm the earlier view which we expressed. The number of complications which arose in them was small, and the only persistent defect noted was deafness. This lamentable condition occurred in a few instances only, and it was more often than not noted early in the disease, before the serum injections were begun."

TUBERCULOUS CEREBROSPINAL MENINGITIS.

Cytodiagnosis. Ribierre and Parturier[1] report a case of tuberculous meningitis with marked polynucleosis and bacillosis of the cerebrospinal fluid. Without being absolutely exceptional, the presence of polymorphonuclear leucocytes in large numbers in tuberculous meningitis, is rare. And those cases in which the tubercle bacilli are found in the cerebrospinal fluid, except occasionally, are few and far between. Indeed, cytodiagnosis teaches that tuberculous meningitis is to be suspected when no bacteria are found and there is a lymphocytosis. It is not necessary to go into the symptomatology of the case, the diagnosis being confirmed by the postmortem examination. The first lumbar puncture made gave issue to an absolutely clear cerebrospinal fluid, though the tension was increased. Examination of the centrifuged specimen showed, nevertheless, large numbers of polymorphonuclear leucocytes, 82 per cent., with 18 per cent. of small lymphocytes. On staining in the usual way for Koch's tubercle bacilli, they were found, hardly a field failing to show less than four or five of them.

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(1) Le Prog. Méd., May 23, 1908.

As to the polynucleosis in tuberculous meningitis, it has been observed in 10 cases in 134 collected by Lutier, and isolated instances have been reported by numerous observers. The polynucleosis is sometimes possibly due to a secondary infection, as in the case of Bruneau and Hawthorn, where the pneumococci were found *with* the tubercle bacilli. It is a fact, however, that when the tubercle bacilli are numerous polynucleosis is present.

The value of examination of the cerebrospinal fluid in the various sorts of meningitis is discussed by Roque and Chalier,[1] their paper being based upon the study of five cases of meningitis or meningism observed by them. In the first case, a girl 20 years of age, always in good health, was found unconscious on a street-car track. An accident was suspected, but except for a well-marked occipital ecchymosis there was no sign of fracture of the skull. There was no paralysis, and no sensory disturbances could be found. There was trismus, a slight contraction of the legs, increased knee-jerks, a suggestion of Kernig's sign, and a tendency for the eyes to roll up. Lumbar puncture showed 95 per cent. lymphocytes and 5 per cent. polynuclear leucocytes. A diagnosis of tuberculous cerebrospinal meningitis was therefore made and confirmed by autopsy. This also showed that there had been a rare sort of fracture of the skull. The tubercles, so abundantly present, had spread from a basilar meningeal tubercle. In this case lumbar puncture led to a remarkable diagnosis. Apparently, say the authors, the meningitis led to the fall and, perhaps, the cranial fracture.

On the other hand, a girl of 17 years of age, with complaints of indigestion, showed also keen headache, occasional vomiting, marked constipation, contractures, stiffness of the neck, Kernig's sign, increased knee-jerks, and delirium. The temperature hovered about 104° F., and the pulse around 100. There was herpes labialis, and a suspicious area of probable tuberculosis in the right pulmonary apex. After a week or so the fever fell and the general condition improved, only to fall back again into the old state. Lumbar puncture, performed rather late, showed clear cerebrospinal fluid, escaping under tension,

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(1) Lyon Méd., July 19, 1908.

not giving the tuberculous sero-reaction, but containing a large number of leucocytes, of which 89 per cent. were lymphocytes, 10 per cent. polynuclear leucocytes, and 1 per cent. endothelial cells. Three weeks later the polynuclears increased to 25 per cent. and the lymphocytes diminished correspondingly. This seemed to rule against tuberculosis. A guinea-pig was inoculated, with negative result. It was concluded that it was a grippal meningitis, and recovery followed, justifying the conclusion. In arriving at this opinion the authors in part were influenced by the presence of herpes labialis. In this case cytodiagnosis at first clearly indicated tuberculosis, whereas later it did not. Three other cases are enumerated by the authors, one being a case of apparent syphilitic meningitis with practically no leucocytes or lymphocytes in the cerebrospinal fluid.

From their observations, the authors regard cytodiagnosis as of varying diagnostic value. Surely the cytologic formulæ are not pathognomonic.

CEREBROSPINAL ARTERIOSCLEROSIS.

Alfred Stengel,[1] in a general paper upon the nervous manifestations of arteriosclerosis, discusses those on the part of the cerebrum. These are various. There is, *first,* the direct relation of arteriosclerosis to thrombosis and hemiplegia or monoplegia. It is possible that other rather indefinite cerebral manifestations are due to hyaline thromboses of the smaller cerebral vessels. Partial thromboses are frequently met with in cases of extensive arteriosclerosis of the circle of Willis and of other cerebral arteries. "These thrombi, though not completely obstructive, cause a rather rapid encroachment on the lumen of the vessels, and thus interfere with the circulation in a manner more abrupt and violent than does the narrowing of the lumen that results from increase of the arterial disease itself. It is not unlikely that the symptoms usually attributed to obstruction of the circulation by increasing arteriosclerosis are often the result of such thrombosis."

A *second* set of manifestations of cerebral arteriosclerosis consists of convulsions of an epileptiform character.

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(1) Am. Jour. Med. Sciences, February, 1908.

Experimental studies show that reduction of the flow of blood through the carotid arteries produces disturbed breathing and heart action, depression of temperature, sleepiness, vertigo, blindness, strabismus, spasms, and convulsions. Indeed, Kussmaul and Tenner cite experiments in which convulsions were readily produced in six rather weak persons. Lucas reports two cases in which elderly individuals with marked arteriosclerosis suffered from attacks of vertigo, headache, twitching of muscles, and great weakness. In one case compression of the carotid arteries caused the rapid development of an attack in which the above symptoms, as well as temporary unconsciousness, occurred. Stengel expresses himself as agreeing with Naunyn that these epileptiform seizures result from cerebral arteriosclerosis, and he recounts the histories of three patients of his own in whom convulsive attacks appeared in arteriosclerotic patients without any other explanation for the seizures. He concludes that "in view of the experimental evidence before cited, it may be regarded as established that sudden interference with the circulation, whether due to thrombosis or embolism, or independent of these accidents, may account for seizures such as occurred in the three cases just described. In the cases in which thrombosis or embolism are absent, weakness of heart action may be the cause of the suddenly developed inadequacy of the cerebral circulation, while in other cases vasomotor spasm may possibly occur."

A *third* set of cerebral manifestations has to do with stupor and coma. The cases included under this group are a little less definitely ascribed to the influence of arterial disease alone, for the reason that the symptoms are less distinctly paroxysmal, and therefore less certainly the result of a temporarily active cause, such as circulatory disturbance. Nevertheless, in view of the fact that both stupor and coma have been produced experimentally by compression of the carotid arteries, and that is some cases the stupor suddenly appears and as suddenly disappears, the connection of the attacks with circulatory disorder does not seem unlikely. If one studies the symptomatology of extreme senility, there will be found a constantly recurring picture of stuporous states alternating with periods of mental alertness often quite surprising. The aged patient without warning falls into a lethargic or semicomatose state, and may remain so for hours or even days, and not infrequently wakes from this condition suddenly and completely, only to relapse again and again. The cause of these attacks may doubtless be an incomplete uremia in some cases, or other forms of toxemia, but the sudden relief of symptoms that so often occurs has convinced the author that the attacks are more usually dependent upon the condition of the cerebral circulation.

A *fourth* set of disturbances comprises attacks of vertigo, temporary aphasia, ocular disturbance, and tinnitus. All these symptoms may be caused by compression of the carotid arteries, or appear among those caused by gross cerebral thrombosis. "When one or the other occurs as a more or less isolated symptom in a case of arteriosclerosis, one hesitates to regard it as due to this cause alone, and very properly inclines to think it a part of some more definite focal, cerebral, or visceral condition not yet fully developed. In many instances further symptoms may occur, and a cardiac, renal, gastric, aural, or local cerebral cause may be recognized. Sometimes, however, such symptoms recur repeatedly and no further developments take place until perhaps with increasing cerebral sclerosis more distinct results follow."

Lastly there are the *psychic* manifestations of arteriosclerosis, to which alienists are giving more attention than they formerly gave to it. Among the distinctive types of mental disease in which arteriosclerosis is recognized as an important pathological lesion are mentioned general paralysis, arteriosclerotic dementia, and, less certainly, senile dementia. Among the special symptoms in the different groups are mental fatigue, headaches, dizzy attacks, memory weakness, psychical phenomena with progressive dementia, fainting, apoplectiform seizures, and epileptiform convulsions. Aphasia, periods of excitement, and mental confusion are characteristic of some. In others the loss of retentive memory may be among the earliest manifestations of the disease. "Later, inclination to fabulate, loss of judgment, disorientation, narrowing of the external interests, episodes of confusion and hallucinatory delirium, follow more or less quickly."

The *treatment* of arteriosclerotic atrophy of the brain is discussed by A. Cramer.[1] After enumerating the prodromata, consisting of headache, vertigo, increasing loss of memory, change in character and individuality, monoplegic, hemiplegic or aphasic phenomena, with possible attacks of late epilepsy and disturbances of consciousness, he gives the organic findings as facial paresis, slowly reacting pupils, anarthria of various degrees, slovenly or delayed speech, amnesic aphasia, transitory hemianopia and ophthalmoplegia, increase of the reflexes, inequalities of the reflexes, sensory paralyses, and occasionally spastic symptoms and disturbances of gait. Associated with these findings are those of the superficial arteries, especially the radial, temporal, and brachial. Often the heart is enlarged toward the left, the tension is increased, and albumin appears in the urine. Occasionally, also, sugar is found. As to occupation, hotel-keepers, actors, officers, bankers, statesmen, etc., especially those subjected more to mental stress and storm, are most frequently assailed.

Treatment resolves itself into prophylaxis and medicinal treatment. Syphilis, the excessive use of alcohol, overeating, and excess in athletics should be avoided by all descending from families prone to early arteriosclerosis. When the disease threatens by its cardinal symptoms of headache, dizziness and weakness of the memory, the brain must be at once put to rest, by removing the patient from his environment, including both his business and his family surroundings; for these usually excite or irritate. Removal from irritants is of paramount importance, and worry must be as completely obviated as possible. Moreover, alcoholic drinks should be forbidden, since these bring about an overaction of the circulatory system. It is likewise important to regulate the diet and to prevent overeating. Bodily exercise must be supervised and excessive exertion vetoed, since such exertion raises the blood-pressure. Moreover, the hydrotherapeutic fads are to be avoided. Instead, merely keep the patient in fresh air, let him take graduated walks and very moderate exercise. It is especially impor-

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(1) **Deut. med. Woch., Nov. 21, 1907.**

tant to watch over the activity of the intestines and to prevent constipation and straining at stool.

As to drugs, Cramer usually prescribes sodic or potassic iodid, in simple aqueous solution. He first gives the patient 15 grains and gradually increases the dose up to 60 grains per diem. In general, it is well not to enforce the taking of too much iodin. In order to do any good the medicine should be given over a long period of time—for a year or more. It may be given intermittently, with occasional pauses of a week or two. During this treatment the heart, stomach, and kidneys must be watched. If the iodid is given in milk, untoward symptoms are not likely to occur. If the appetite is lost and albuminuria appears, iodipin may be given. If conditions of mental confusion and disorientation occur, bromids should be added. This is preëminently true in arteriosclerotic senile epilepsy.

CEREBRAL ANEURYSM.

Cecil F. Beadles,[1] in a lecture delivered at the Royal College of Surgeons of England on aneurysms of the larger cerebral arteries, based upon an analysis of 555 cases, asserts that in 257 the first symptoms were those of apoplexy. In 115, signs suggestive of a tumor had been present before the fatal apoplectic stroke, but in very few of these was a tumor actually diagnosed. In 91 cases there was no cerebral or meningeal hemorrhage, but signs and symptoms of a brain tumor or lesion of some kind had been present. In most of the remaining 92 cases no mention is made of any clinical condition referable to an aneurysm. In 78 there were signs of compression of one or more cranial nerves. In 339 of the cases death was due to rupture of the aneurysmal sac. Beadles reports many cases and discusses numerous pathologic specimens from various pathologic museums. He concludes as follows: "Notwithstanding the remarks made by some who have specially written on intracranial aneurysms, and the writers of some of our leading text-books, the conclusion that I have been forced to draw from a careful study of a large series of cases is that it is quite impossible to diagnose an aneurysm of any one of the cerebral arteries except

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(1) Brain, Part III, 1907.

in the most unusual circumstances. Only two or three have ever been diagnosed during life, and even in these cases it can scarcely be said to have been an absolutely certain diagnosis.

"I will go further and say that in the vast majority of cases of aneurysm a tumor even cannot be diagnosed. I have mentioned a number of interesting cases which presented symptoms, one might say, clearly pointing to an intracranial tumor. But these, I would point out, were exceptional cases. They are in the minority. With the majority there have been no distinguishing signs or symptoms. Usually when they occurred they might readily be produced by other lesions than those of a tumor. In many cases there has been an entire absence of symptoms, sometimes when the aneurysm has been of considerable size. The apoplectic symptoms following rupture of an aneurysmal sac are indistinguishable from those of cerebral or meningeal hemorrhage from other causes.

"If there is any one sign to which special attention might be drawn, it is the occasional intermittent character of the symptoms. But these form a very small proportion even of the cases where tumor symptoms were present, and, after all, it is not very uncommon for symptoms to show variability in intensity, or even a temporary disappearance, in some other forms of tumor and in other cerebral diseases.

"The impossibility of diagnosing a cerebral aneurysm by those who have recorded cases cannot be placed to the ignorance of the observers. Many of the cases have been under the direct observation of the most learned physicians of the day, both in this country and abroad. The exact diagnosis of a cerebral aneurysm during life is no more possible today than it was when Gull wrote, nearly fifty years ago: 'Although we may from the circumstances sometimes suspect the presence of aneurysm within the cranium, we have, at the least, no symptoms upon which to ground more than a possible diagnosis.' "

A. Souques[1] reports a very interesting case of a large aneurysm of a branch of the middle cerebral artery, diagnosed in life as a benign cerebral tumor. There was parox-

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(1) Nouvelle Iconocle la Solpêtrière, March-April, 1908.

ysmal headache, nausea, and vomiting, vertiginous attacks, epileptiform and apoplectiform seizures. The tumor was located in the right Rolandic area, because of the headache and the left hemiplegia. The patient committed suicide by jumping from an iron stairway, and, to the surprise of all concerned, an aneurysm was found. Had a diagnosis been made, operation would have been of no avail.

Cecil F. Beadles[1] describes a patient presenting the clinical symptoms of general paralysis of the insane, in a man of 38 years of age, in whose brain were found multiple small aneurysmal dilatations on the larger arteries at the base, affected with syphilitic endarteritis, syphilitic deposits and softenings in both cerebral hemispheres, and also presenting symmetrical aneurysms at the bifurcation of both common carotid arteries. There was no seizure immediately preceding his death, though he had several during the course of his illness. Throughout the whole time he was under observation no paralysis of either limbs or face was observed. The eyes presented no abnormality beyond the inequality of the pupils and their feeble reaction to light. No defect of any of the special senses was detected. The patient was melancholic, slow to reply to questions, dull, and very reserved. His memory was impaired, and toward the end delusions of grandeur appeared. Of interest is the fact that during life the patient presented the picture of general paralysis of the insane, although after death multiple aneurysms and syphilitic deposits and presumably softened gummata in the cerebral hemispheres were found. The presence of such gross syphilitic lesions cannot be said to nullify the clinical diagnosis of general paralysis, according to Beadles. Moreover, in this case the numerous dilatations in the basal arteries were all syphilitic, and tend to affirm the doctrine that intracranial aneurysms are usually specific.

CEREBROSPINAL SYPHILIS.

F. W. Mott, in an address on recent developments in our knowledge of syphilis in relation to diseases of the nervous system,[2] first calls attention to the undoubted variability in the virulence and toxicity of the various

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(1) Edinburgh Med. Jour., August, 1907.
(2) Brit. Med. Jour., Jan. 4, 1908.

strains of the *Spirochæta pallida,* and suggests that possibly there may be a certain strain which produces a special neurotoxin.

"To sum up, then, the specific virus of syphilis is probably a protozoon, some forms of which may be attenuated in their virulence owing to the passage of the organism through the bodies of certain individuals; while other forms may be especially virulent for the same reason, and acquire a special neurotoxic action. We are, however, on much more certain ground in discussing the variation of the symptom-complex which will follow infection, when we refer it to the vital resistance of the individual himself; and we may represent this in the form of an equation, thus:

$$\text{Symptom complex } x = \frac{V}{R} = \frac{\text{virus}}{\text{resistance}}$$

But R is made up of a number of factors, some of which we can ascertain, but it is generally impossible to decompose R into all its constituents. Roughly speaking, we may say that it is made up of what a man is born with, what has happened after birth, and what will happen in the future to resist the action of the specific virus, which in the majority of instances is of lifelong duration."

Mott believes that syphilis is the essential cause of tabes and general paralysis, and he enumerates the following facts to support his belief:

"1. The remarkable statistics of Erb.

"2. Very few eminent neurologists or psychiatrists now believe otherwise—*vide* Dr. Ferrier's Lumleian lectures.

"3. The cerebrospinal fluid of tabes, general paralysis, and syphilitic meningitis invariably contains lymphocytes, often plasma cells, and no polymorphonuclears. This condition practically occurs in no other *chronic affection* of the nervous system, excepting sleeping sickness, and in this respect it affords another important analogy.

"4. The recent work of Morgenroth, Wassermann, Plaut, Stertz, Marie, and Levaditi, by Bordet and Gengou's method, shows the existence of antisyphilitic bodies in the serum and cerebrospinal fluid of tabes and general paralysis, the quantity increasing in amount as the disease progresses.

"5. The experiments of Krafft-Ebing. He noticed, as most observers have done, that, in spite of the promiscuous sexual intercourse of paralytics in the early stages of their affection, a primary sore, or even secondary eruptions, were extraordinarily rare. He was convinced that the reason was that they had an immunity from previous inherited or acquired infection. Consequently nine advanced general paralytics, without any signs of syphilis on the body, and with no history of the same, were inoculated with the virus of a hard chancre and watched 180 days. Not one exhibited any signs of the disease.

"6. It is said that sexual excesses, alcoholism, and all those conditions of town life which lead to neurasthenia are essential, and that syphilis cannot of itself produce the decay of the nervous system. Very true is this statement, if it means that syphilis is the necessary basis, and that these are factors which increase the liability to a destruction of the metabolic equilibrium of the neurones and cause their premature decay.

"7. In 80 per cent. of 40 cases of juvenile general paralysis I found syphilitic antecedents were determinable and the influence of congenital syphilis firmly established; in the remaining cases, either no history was obtainable or syphilis could not be excluded. Now, alcohol, sexual excesses, and the conditions leading to neurasthenia, *except those due to inheritance,* could not play an active part.

"8. The Argyll-Robertson pupil and the irregular pupil are practically only met with in general paralysis, tabes, and syphilis; it may be the sole sign of syphilis.

"9. Whereas the proportion of males to females affected by tabes and general paralysis depends upon the social status—that is, the liability to syphilis—in the juvenile cases, where the chances of infection are equal, one does not find this difference of the sexes.

"Parasyphilitic disease of the nervous system depends upon two factors, intrinsic, innate, and extrinsic, acquired —the soil and the seed; the vital resistance and the specificity of the virus, $\frac{V}{R}$. All those conditions, which may be inherited or acquired, and which tend to active metabolism of systems, communities, and groups of neurones functionally correlated, and which, owing to those conditions of stress which in one individual would cause spinal neurasthenia, in another cerebral neurasthenia, will, in conjunction with the stimulating effect of the syphilitic poison, cause the nerve cells to exercise an abnormal metabolic activity in the production of the side-chain molecules necessary for immunization against the toxic effects of the virus.

"We know that one attack of syphilis confers immunity during the rest of the individual's life, and the experiments of Krafft-Ebing are important to remember in this respect. The nerve elements being perpetual, having acquired a habit of throwing off side-chain molecules, will continue to do so during life and will contribute largely to the immunity produced. When there is no longer metabolic equilibrium, and decay sets in, these immune bodies are thrown off in increasing numbers; this seems probable from the fact that in general paralysis and tabes the quantities increase with the progress of the decay. The process of decay will manifest itself in the earliest stages by an increased irritability and functional activity of the nervous structures, often manifesting itself in a *hyperesthesia sexualis,* and not infrequently in striking intellectual activity, followed in each case by exhaustion and loss of function.

"The uselessness of antisyphilitic remedies is thus easily accounted for; indeed, they are generally positively injurious in true tabes and general paralysis, because they lower the vital energy in a system which has overimmunized itself against the syphilitic virus. The only hope of doing any good is by an early diagnosis of the disease and suppression of all those exciting causes which use up the nervous energy and tend to overturn the metabolic equilibrium of the central nervous system, causing its premature decay. This may explain a well-known fact, first pointed out by Benedikt, that tabetic patients who become blind from optic atrophy remain in the preataxic stage a great number of years. The loss of sight necessitates mental inactivity, provided the patient does not worry."

Diagnosis and Treatment. T. H. Weisenburg[1] discusses the diagnosis, and treatment of syphilitic lesions of the nervous system. Very interesting are the questions as to why one person has the nervous system affected after the acquisition of a specific infection, while another has not. It is probable that the most important factor which determines whether the infected person will have future involvement of the nervous system is the lessened resistance of these structures, or possibly a special predisposition. Hereditary and family influences surely have a bearing upon the question. It is largely a matter of conjecture as to the percentage of cases of syphilis subsequently suffering from disease of the central nervous system. Erb places the incidence of tabes between 1 and 5 per cent. of all persons affected with syphilis. Weisenburg conjectures that from 5 to 10 in every 100 individuals with syphilis show disease of the nervous system. It is a question whether or not early treatment in the disease forfends disease of the nervous system later on. "That it does so in the majority of cases, there is no doubt; but in many instances it is of no avail." Two examples are given of acute disease of the nervous system occurring in the midst of very vigorous treatment, and they tend to show the uselessness of treatment in certain forms of syphilis. The conclusion which Weisenburg reaches is that "In some forms of syphilis the cerebrospinal system will become diseased whether or not treatment is instituted, and no matter how vigorously."

"After an infection the nervous system may be involved almost immediately, even in the midst of treatment, or may not be involved for some years, sometimes as many as twenty. It is generally supposed that the earlier the cerebrospinal symptoms appear, the more severe the infection. Considering, however, that the symptoms produced years after the infection are about of the same severity and character as they are in the early periods, there is probably little difference."

(a) The early forms of cerebrospinal syphilis are several. (1) Among the earliest is a myelitis, usually of sudden onset, and in most instances affecting the lower

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(1) Am. Jour. Med. Sciences, April, 1908.

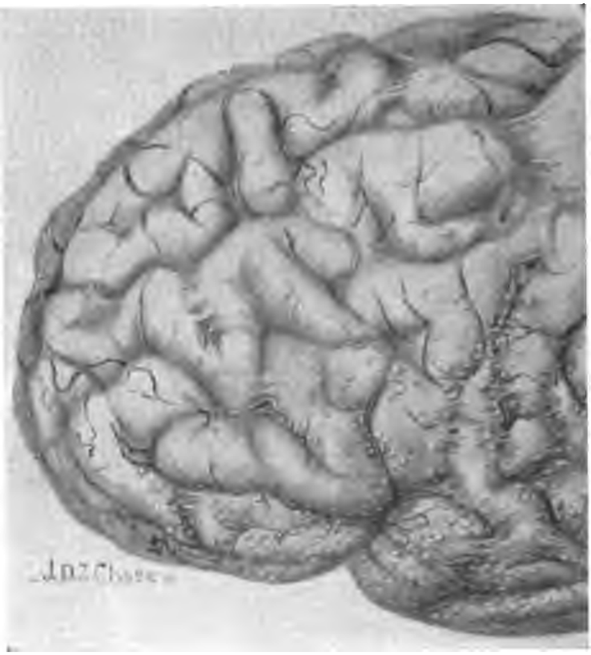

PLATE I.

Left prefrontal lobe, showing miliary carcinomata and their relation to the cerebral veins.—D. J. McCarthy and M. K. Meyers (page 65).

thoracic or thoracic-lumbar region, there being loss of power in both lower limbs, of the bladder and rectal functions, and of sensation. Added to the symptoms of the myelitis there will be those of meningeal involvement, such as girdle-sense and pain. (2) Other common early manifestations, sometimes appearing in the midst of treatment, are the result of multiple lesions of the brain and cord, giving diffuse symptoms, both motor and sensory, of irregular character and distribution. (3) Hemiplegia in early syphilis is common, and nearly all hemiplegias occurring before 40 years of age are syphilitic. (4) Basal meningitis is nearly always greatest around the chiasm, and hence involves the optic, oculomotor, and abducens nerves. (5) Oculomotor paralysis, unilateral in type, has been termed the *sign manual* of syphilis.

(b) The late forms of cerebrospinal syphilis, those occurring five years or more after infection, usually preponderate either in the brain or in the spinal cord, but not in both places at once. "When the brain is mostly involved there will be, besides the irregularity of the pupils and the slowness of reactions to light and movement, other symptoms depending upon the location of the lesion. These may consist of a gummatous deposit in almost any portion of the brain or of a basal infiltration, besides those diseases resulting from the early endarteritis." Three cases in illustration are presented.

Syphilis may sometimes cause the general symptoms of brain tumor without any special localizing signs. Headache, nausea, vomiting, and bilateral choked disk may alone appear, with perhaps irregularity of pupils and slow pupillary reactions. "In those instances in which the spinal cord is mostly involved, there may be very diffuse symptoms, but in nearly all there is some meningitis, thus causing girdle-sense and pains." Cases are observed in which the involvement of the brain and the spinal cord is equal. The symptoms will, as a rule, be multiple, and there will be, besides the specific pupillary phenomena, disturbance of mentality, irregular motor and sensory symptoms, and involvement of the bladder and rectum.

Treatment. "It is in the early stages of this disease, in the period of round-cell infiltration, that antispecific treatment is of the greatest benefit, and treatment should be promptly and vigorously instituted. The best results can be obtained from inunctions of mercury, a dram a day, to be rubbed in in different places. The best plan to follow is to apply this daily, with the exception of Sunday, and to keep it up for a period of about six weeks; if any improvement can be hoped for, it will be manifest in that time.

"It has been my experience that whenever any benefit has been obtained, it has been the result of the use of mercury, and not of iodids. Even in the specific manifestations which come on many years after the infection, mercury is the only drug which will be of benefit. Iodids probably do good, and should be given either in conjunction with the mercury or, better still, after the mercurial treatment. It is a good plan to give them in increasing doses, 50 to 70 grains a day being quite enough."

Paul Schuster[1] discusses the general question as to whether the *mercurial treatment* of syphilis has any influence upon the subsequent development of the so-called metasyphilitic diseases, especially tabes and general paralysis. He first reviews the work of Wassermann, Plaut, Neisser and others on the syphilitic antibodies found in the blood serum and cerebrospinal fluid of old tabetics and paralytics, and then studies the question statistically, quoting especially figures from Neisser, Eulenberg, and Dinkler, and adding 75 cases of tabes, 35 of general paralysis, 76 with cerebrospinal syphilis, and 24 with ulcer, of his own. As a result of this study he concludes as follows:

"(1) The clinical average picture of tabes and general paralysis of the insane is the same whether the patient were treated with mercury or not (2) The metasyphilitic nervous diseases appear in patients treated with mercury no later than they do in patients not so treated. (3) There is no proof that treatment with mercury exerts a favorable influence with reference to the avoidance of subsequent nervous diseases. (4) Many facts resulting from serologic investigation appear to demonstrate the fact that the metasyphilitic nervous diseases are not caused by the syphilitic virus, but are instead brought into being by the syphilitic antibodies."

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(1) Deut. med. Woch., Dec. 12, 1908.

Prophylaxis. Debove, in a general article on multiple syphilitic lesions in the central nervous system,[1] after reporting a case of tabes, general paralysis and aortic insufficiency in a syphilitic, says that after coitus with an individual suspected to be infected, vigorous rubbing of the glans penis with an ointment containing one-third calomel and two-thirds lanolin may prevent the acquisition of the disease. In winter it is advantageous to add one-tenth part of vaselin. According to Roux and Metchnikoff this statement has been tested out upon a monkey and also upon a physician inoculated first with the virus. This ointment, however, protects only during a few hours after contact, and it is questionable whether it will ever succeed in diminishing syphilis, even if generally adopted.

Atoxyl acts at a period when the mercurial ointment ceases to be effective. Experiments by Roux and Salmon made upon the monkey demonstrate that atoxyl may arrest syphilis during the period of incubation. Some monkeys have had the syphilis aborted by injections of atoxyl made 2 weeks after inoculation. They have then been reinfected, and a chancre has been produced, thus proving that no immunity had been conferred by the first inoculation. At present we do not know exactly how long atoxyl has an aborting influence, but it is probably only a certain number of days. The dose for an adult, used in this way, is 50 centigrams in 5 grams of water, to be given hypodermically and repeated in 2 days. Two such doses are sufficient. Unfortunately a man does not have intercourse with a woman he suspects to be syphilitic, and hence he is always caught napping.

CEREBRAL ABSCESS.

Etiology. E. B. Dench[2] discusses the subject of brain abscess from the point of view of the otologist, while reporting 2 fatal cases, one being located in the cerebellum and the other in the cerebrum. The cerebral case followed the lighting up of a 10-year-old otitis media by an attack

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(1) Gaz. des Hôp., Feb. 20, 1908.
(2) Am. Jour. Med. Sciences, November, 1907.

of grip. Mastoid tenderness was present even after incision of the ear-drum, so 48 hours later a mastoid operation was performed. The patient improved for 6 weeks, at which time the mastoid wound had almost entirely healed, and the temperature had long since fallen to the normal. A sudden elevation of temperature followed, with a leucocyte count of 11,400 and 84 per cent. of polymorphonuclear cells. Thrombosis of the external anterior frontal branch of the middle cerebral artery was suspected by several physicians, and at their advice operation was not performed. The symptoms growing worse, and the leucocyte count constantly mounting, a chill supervening, and headache appearing, the skull was opened and a very large abscess found in the left inferior frontal convolution and the adjacent island of Reil.

Of 100 cases collected by Dench and Chas. E. Perkins, 20 followed an acute otitis media, 77 followed a chronic suppuration, and in 3 the character of the otitis is not stated. The route of infection was through the tegmen tympani in 40 cases, secondary to epidural abscess in 6, to sinus thrombosis in 6, through the mastoid antrum in 6, and through the squama in 2. In the other cases either no bone defect was found or no statement was made as to route of infection.

As to *symptoms,* 77 complained of headache and 44 of vomiting. The pulse was slow in 37, there was vertigo in 32, coma and stupor in 31, mental dulness in 20, aphasia in 10. Optic neuritis was present in 32, absent in 20, and not stated in 48. Other symptoms noted were general convulsions, pupillary disturbances, and exaggerated patellar reflexes.

The results of *operative treatment* were gratifying, 52 recovering and 48 being fatal. In 41 the abscess was opened through the tegmen tympani, and 27 recovered. Of 37 opened through the squama, 18 recovered. If the abscess can be opened along the avenue of infection there is little danger of secondary meningitis or of hernia cerebri. If the abscess is opened through healthy dura the danger of meningitis, on the contrary, is very great. In these cases it is best to do a two-step operation, opening the skull, incising the dura, and packing with iodoform gauze, deferring the exploratory incision of the brain for from 12 to 24 hours. Unfortunately delay is not often possible.

Clinical Cases. A. A. Gray[1] reports a case of cerebral abscess interesting on account of the unusual course which it ran, and the peculiar conditions found postmortem. The child, 7 years of age, had a chronic discharge from the right ear, dating from shortly after birth. Three weeks before admission to the infirmary she caught a cold, and soon complained of pain in the right ear and across the brow; and at the same time the discharge became more profuse. Three days before admission the discharge suddenly ceased and the pain became worse. The day before admission the face was noticed to be twisted toward the *right* side, and the patient was unable to open the right eye. There was no chill, vomiting, twitching or convulsion. Examination showed a well-marked left facial paralysis, ptosis of the right eyelid, associated with external strabismus, and dilation of the pupil, and pus in *both* ears. In view of the fact that the history showed the right ear to be the chief offender, the right antrum was opened, but no opening into the cranial cavity was found. The lateral sinus was found healthy. The dura was then incised over the roof of the antrum, and a tenotomy knife was passed vertically upward only 2 to 3 millimeters. At once a profuse quantity of extremely foul-smelling, opaque but watery, fluid was discharged. From this it was inferred that the abscess had already broken into the lateral ventricle, which later was found to be correct. An iodoform gauze drain was inserted, and curiously the patient began to improve. Ultimately she again grew worse and died about 2 weeks after the operation.

Postmortem examination showed pus in both lateral ventricles, and in the third and fourth ventricles. A minute area of carious bone was found on the upper, posterior and internal wall of the antrum, and a probe could be pushed through into the cranial cavity. The chief interest lies in the rupture of the abscess into the lateral ventricle, which caused the partial loss of consciousness. Strange to say, such rupture was not immediately fatal, perhaps because there was merely a gradual leaking through

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(1) Glasgow Med. Jour., June, 1908.

of the pus. From the lateral ventricle the pus spread through the foramen of Monro to the third ventricle, back through the aqueduct to the fourth ventricle, and through the foramen of Magendie to the arachnoid space, where it finally set up a purulent meningitis involving almost all of the cranial nerves and entailing a fatal result.

R. E. Coughlin[1] reports a case of cerebral abscess with *marked symptoms*. "Briefly stated, this was a case of a young woman with a strong family history of tuberculosis; a history of pain in her right ear without any discharge of pus 2 years previously; pain in the right occipital and parietal region coming on 2 months before her admission to the hospital, this pain being constant and unrelieved by treatment and worse at night; forcible vomiting; vertigo; anæmia; a leucocytosis of 18,000; a low tension pulse; patient dying after all apparent means had been employed to locate her disease; and the necropsy showing the diseased condition in the left hemisphere of the brain, where there had never been the slightest sign to indicate the location of the abscess in this situation. Had the consulting surgeon operated over the site of trouble as indicated by the symptoms he never would have reached the diseased area.

Operative evacuation of the abscess is the only treatment that can be considered, yet how impossible it is to operate when the symptoms bear no relation to the location of the abscess, as in this particular case. Without such an operation the disease is, of course, fatal.

Necropsy: Upon opening the calvarium an irregular, burrowing abscess in the left hemisphere just external to the lateral ventricle could be made out. This burrowing pus could be traced to a small opening entering the right ear. The base of brain was covered with exudate. A complete postmortem examination was not performed, by request of the family."

Foci of Softening in Bright's Disease.

Paul Courmont and A. Cade[2] report a case from Teissier's service, of a baker, 64 years of age, suffering from

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(1) New York Med. Jour., April 11, 1908.
(2) Lyon Med., May 24, 1908.

Bright's disease. Examination of the eyes showed a remarkable limitation of the field of vision, with the preservation of a small triangle for each eye in the lower quadrant. The retina showed gray atrophy especially marked in the left eye. Associated with these findings were diminished mental activity, impairment of speech, attacks of vertigo, and paresthesiæ on the right side of the body. There was word-deafness, jargonaphasia, and echolalia. Word-blindness, and writing could not be tested because of the condition of his eyes. A year later he died, and there were found postmortem multiple lesions of cerebral softening, predominating in the left hemisphere. In this latter were found foci in the posterior part of the lingual lobule, the back part of the fusiform gyrus, the anterior portion of the lingual gyrus, and the mid-portion of the gyrus fusiformis; a few foci in the third occipital, in the third temporal, in the anterior portion of the first temporal gyri, and in the parietal lobe behind and above the end of the fissure of Sylvius, in the island of Reil, and finally a focus in the ascending parietal convolution. All these foci of softening, almost as widely scattered over the right hemisphere, were superficial and scarcely involved the subjacent white substance. In the interior of the brain was a focus of softening in the front end of the left lenticular nucleus. The authors ask the question whether the foci of softening in the occipital lobe can account for the retinal changes. They answer the question negatively [just as it always has been answered—Ed.], calling attention to the development of the clinical portion of the case, which alone precludes an affirmative answer. Microscopic examinations of the optic pathways from retinæ to occipital lobes, were not, however, made.

Brain Trauma.

G. B. Buchanan[1] reports a case of injury to the motor area of the brain, in which a man was hit on the head by a block of wood measuring about 1 ft. square and 3 ft. in length, falling from a height of 25 or 30 ft. It naturally caused a compound fracture of the skull. The patient was knocked down insensible, but soon recovered

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(1) Glasgow Med. Jour., March, 1908.

partial consciousness. Examination showed a compound comminuted fracture of the skull, some of the fragments being depressed and partly under the edges of the skull opening. "In addition, a piece of wood about 2 fingers-breadth in width and about a quarter of an inch thick was embedded in the brain." The wound bled profusely, and there was a large amount of effusion around it. The patient was dazed, but not entirely unconscious. He was unable to move the limbs on the left side, and on that side there was facial paralysis.

A month after the operation the wound was closing by granulations, but on account of the continued discharge of necrotic brain tissue in small quantities the sinus was kept open with a narrow tube. About 7 weeks after the accident the sinus had closed, and 2 weeks later the patient left the hospital. By this time "the paralysis of the face was only slightly apparent. The leg was much stronger, but still very weak, and the patient could not walk without a great deal of assistance. The left arm was much thinner than the right; there was slight movement in it, but the limb was practically useless."

Five months after the accident "the patient was examined at his own home. For the last 2 months he had been more or less confined to bed. He was at times very restless and obstreperous, and, as a rule, particularly so at nights, and required fairly large doses of bromids to quiet him. He was inclined to be sleepless, and was easily irritated by even slight noises, and especially by the children playing outside and people working at a dock near at hand. His general functions remained satisfactory, however, the bowels acting regularly, and urination being normal.

"There was no apparent interference with the eye mechanism, and the eyelids could be opened and shut. The pupils were equal and reacted to light. The brows could be raised, depressed, and wrinkled apparently almost normally. There was slight flattening of the left side of the face, and the actions of the levator and depressor anguli oris were weaker than on the right side." He could not whistle, and saliva dribbled from the left corner of the mouth. The right limbs could be freely moved.

"The chief points of interest in the case are:

"1. The small amount of damage done by such a missile falling 25 to 30 ft. 2. The absence of suppuration notwithstanding the dirtiness of the wound and the continued loss of brain substance. 3. The effect of removing surface irritation in relieving the restlessness and insomnia. 4. The recovery of certain parts formerly paralyzed, just as in cases of cerebral hemorrhage followed by rigidity, and the absence of Jacksonian attacks in spite of the extensive cicatricial process."

HEMIPLEGIA.

Hemiplegia with Contralateral Optic Atrophy. R. T. Williamson[1] reports 4 cases of hemiplegia with contralateral optic atrophy. These cases are rare, but easily explained. The hemiplegia is due to thrombus of the middle cerebral artery, and the optic atrophy to extension of the thrombus downward into the internal carotid to the origin of the ophthalmic artery, which would thus be deprived of blood. Thus, for example, if the thrombus were in the left middle cerebral artery, there would be a right-sided hemiplegia and a left-sided optic atrophy—precisely what actually occurs. This explanation was confirmed by a postmortem examination had in the fourth case.

"In this case the pathological examination could leave little doubt that the optic atrophy on the *right* side, with hemiplegia on the *left* side, were due to thrombosis of the *right* internal carotid extending to the middle cerebral, and also causing occlusion of the central artery of the retina on the right side (either by thrombosis which had extended along the ophthalmic artery to the central artery of the retina, or by an embolus detached from a clot in the internal carotid which had passed along the ophthalmic artery to the central artery of the retina).

"These cases are of interest with respect to the diagnosis and localization of the lesion. Probably a number of similar cases have been recorded in medical literature, but I believe that this combination of symptoms and its cause are not well known to medical practitioners."

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(1) Brit. Med. Jour., June 6, 1908.

POLIOENCEPHALITIS SUPERIOR.

W. A. Holden and J. Collins report 6 cases of polioencephalitis superior, and discuss its cause, clinical course and termination.[1] "It is now generally believed that the morbid condition constituting the anatomic basis of this disease is not unlike that constituting the pathologic process in various other inflammations of the gray matter throughout the central nervous system. In other words, the anatomic basis of acute, hemorrhagic, non-purulent encephalitis may be practically similar to that of acute superior polioencephalitis, and the different course and termination of the latter is conditioned particularly by the location of the disease and by the proximity of important structures which may become secondarily disordered. The picture of the pathologic process that one has in mind when the diagnosis of acute hemorrhagic encephalitis or acute bulbar or spinal poliomyelitis is made will adequately suffice for the pathologic process in acute polioencephalitis superior.

"When the inflammation consisting of superior polioencephalitis confines itself narrowly to the gray matter of the third ventricle the symptoms are few and distinctive, as they are in anterior poliomyelitis, and they can scarcely be mistaken for those dependent upon any other disease. It is only when the inflammatory process extends beyond the aqueduct of Sylvius to the fourth ventricle, as it sometimes does, that the clinical picture becomes more complicated. In some instances it extends through the pons, the cerebellar peduncles and the basal ganglia in a most irregular way, and in such cases the resulting symptoms are so disparate that a satisfactory diagnosis can scarcely be made.

"*Etiology and Pathology.* In regard to the etiologic and pathologic contributions to our knowledge of the disease, the most important are, first, that the same causes that are responsible for inflammation of the gray matter in other parts of the central nervous system are responsible for this disease. Acute infections, such as influenza, pneumonia and diphtheria, have all been accused of being the cause of the disease and a very strong case made out against them. It is not only infections, however, in whose

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(1) Jour. Am. Med. Assoc., Feb. 15, 1908.

wake it follows, for cases have been reported which seemed to show that lead, oxid of carbon, sulphuric acid, etc., were responsible factors.

"Study of the central nervous system of many of these cases in which a postmortem was held has shown that in a number of them the structural changes are slight and entirely out of proportion to the severity of the symptoms that existed. Indeed, in some instances no lesion adequate to account for the symptoms or the fatal termination of the disease was found. These cases are looked on as analogues of asthenic bulbar paralysis (bulbar paralysis without anatomic foundation), in which it is supposed that several levels of the different segments of the central nervous system are so overwhelmed by some toxic agent that they become functionless to a certain degree, and in some instances wholly so. It is supposed that the injurious agency, whatever it may be, is not of sufficient severity to cause discernible morbid changes in the cells.

"*Symptomatology.* The typical clinical manifestations of acute polioencephalitis superior fall into 3 distinct groups: those referable to the eyes, the mental disturbances, and the ataxia of gait and speech. The symptoms usually develop slowly, preceded by vertigo, headache, diplopia and sleeplessness, although they may come most abruptly, the eye-muscle paralysis being very sudden. The ophthalmoplegia, usually spoken of as exterior because it affects only the extrinsic muscles of the eye, may, however, exceptionally be manifest by paralysis of the ciliary muscle and the sphincter of the iris. In the 7 cases the ophthalmoscopic examination reveals an optic neuritis, the paralysis of the external eye muscles, the frequent partial ptosis, and the peculiar whimpering, pathetic facial expression which the patient develops, constituting a very typical clinical picture. The mental symptoms are usually those of an active delirium resembling that of alcoholism, and likewise the ataxia of speech and of gait is not unlike that of acute inebriation. Occasionally the mental symptoms are quite like those of Karsakow's syndrome, with disorientation, spurious reminiscences, active fabrication, particularly following suggestion, and easily provoked mental confusion."

The cases here reported show that the disease terminates in recovery much more frequently than is generally taught.

"Spiehlmeyer says that only those cases which have shown 3 cardinal symptoms, including grave affection of the sensorium, delirium or somnolence, should be included, and our cases show these symptoms. He maintains that the majority of patients that are classified as recovered keep certain mental symptoms, which he calls chronic delirium, and this should make us hesitate to classify them as cured. But our negro woman, for instance, did not have any chronic delirium during the months she was in the hospital under the constant observation of one of us. Her case we regard as an important contribution to the literature, because it shows that cases of polioencephalitis developing from alcohol may recover. In this respect it corroborates Boedeker, who reports the case of a university professor who had a typical attack of polioencephalitis superior due to the intemperate use of alcohol, who made a complete recovery and resumed his professional duties."

CEREBRAL TUMOR.

Symptomatology. The symptomatology of brain tumors is discussed by Hans Hirschfeld,[1] who reports 2 cases. The diagnosis is always twofold: the general diagnosis of tumor and its localization. The first case was a large adenocarcinoma compressing the right cerebellar hemisphere, and was accompanied by 3 other similar tumors, 1 being extracerebral and the other 2 intracerebral. The patient, a woman 63 years of age, began to suffer from headaches in the spring of 1906. By November intelligence was slightly affected, and a beginning choked disk was found, with Romberg's sign slightly developed. The pulse was 80, but in an attack of severe headache, with a congested face, fell to 48. Later the pulse became persistently slow, 40 to 50 beats, and the pupils ceased to react. A diagnosis of occipital tumor was made, but operation was not permitted. At the postmortem the above tumors were found.

A second case illustrates the difficulty of diagnosis. The patient, a woman 36 years of age, began to have headaches, which were supposed to be migraine, her mother having had sick headaches for years. The pupils were

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(1) Berl. klin. Woch., Dec. 30, 1907.

fixed, but the eye-grounds were normal. The knee-jerks were absent, the pulse 40. There was vomiting. No other nervous symptoms presented. Lumbar puncture was forbidden by the family. Energetic specific treatment cleared up everything in 4 months, restored the pupillary and patellar reflexes. After 2 weeks of calm the patient suddenly lost her speech, which motor aphasia disappeared in 3 weeks. A little while later she fell dead. Postmortem examination showed a tumor of the hypophysis, aneurysm of the carotid artery, and proliferating endarteritis of the smaller vessels. No change was found in the optic nerves. The signs of tumor, headache, vomiting and bradycardia are attributed by the author to the aneurysm of the internal carotid artery. Parietal thrombi forming in the walls of the aneurysm produced an embolus which caused the sudden motor aphasia. The small tumor of the hypophysis apparently played no part in the symptomatology. Surely in this case a localizing diagnosis was hardly possible.

J. L. Atlee and C. K. Mills[1] report a case of brain tumor with Jacksonian spasm and unilateral paralysis of the vocal cord, late hemiparesis and astereognosis. The patient, a physician, 43 years of age, first noted hoarseness in March, 1905. In September he had numbness in the left lips, face, neck, and throat. In June, 1906, he was taken with a cramp or spasm in the left foot and leg. In a few minutes the spasm extended up the leg and body until it involved the arm. He then fell unconscious, remaining so about 20 minutes. After regaining consciousness he was simply confused; he did not vomit, had no nausea, and did not pass into a sleep or stupor. Eight or 9 weeks later he had a similar attack, and had time to lie down before becoming unconscious. His facial attacks were mere spells of numbness, with inability to speak. In September, 1906, when examined, no extra-ocular palsies were present, and no paralysis could be determined in the distribution of either the fifth or seventh nerves, although the naso-labial fold seemed to be deeper on the left than on the right. Paralysis or paresis was entirely absent in the limbs. The deep reflexes, both in the upper and lower

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(1) Jour. Am. Med. Assoc., Dec. 28, 1907.

extremities, were prompt, but more so on the left. Ankle clonus was absent on each side. Sensation for touch, pain, and temperature was normal all over the body, as was also the sense of location. With the hand absolutely relaxed, the patient did not seem to recognize when the left thumb and forefinger were moved. Astereognosis was not present.

Ophthalmoscopic examination showed normal vision and no optic neuritis. In December weakness appeared in the left side, and there was failure of word memory, both in speech and writing. In the latter part of January, 1907, the weakness on the left side was marked, the left deep reflexes were accentuated, and there was astereognosis on the left side and failure of word memory. The voice was practically entirely lost.

The diagnosis of a tumor of the cerebrum, originating at the lower end of the precentral convolution and involving a large part of this convolution, and to some extent the parietal lobe as shown by the presence of astereognosis, was now considered practically certain, in spite of the absence of the general symptoms with the exception of recent headache, and it was decided to operate.

The tumor was found to be correctly localized, and was removed. It measured 2½ by 2 by 1¾ inches. Examination of the tumor by W. G. Spiller showed it to be epithelial in character, doubtless springing from the choroid plexus.

"Whether or not the unilateral paralysis of the vocal cord was due to the cerebral lesion is a question more interesting and much more difficult. This paralysis, as determined by the study of the patient's condition and also by laryngeal examinations, had all the features of a unilateral peripheral recurrent laryngeal affection, and, indeed, it cannot be positively stated that it was not due to such peripheral disease. It is interesting, however, to recall that the patient's voice, which had disappeared entirely, recovered considerably after the removal of the cerebral tumor, although at the time the last laryngeal examination was made no great change had taken place in the paralyzed and somewhat atrophied vocal cord. It is generally believed that the cortical laryngeal centers have bilateral control, in other words, that the center in each hemisphere controls the movements of adduction and abduction of the vocal cords on both sides."

Unusual Tumors. (a) *Hemicraniosis.* This is a term applied by Brissaud to hyperostosis of the bones of half of the cranium, including the face.[1] W. G. Spiller,[2] after calling attention to the only other cases in the literature, reported by Parhon and Goldstein, and by Parhon and Nadjede, reports 2 cases, both showing enlargement on the left side, in one case the enlargement being strictly unilateral and in the other slightly involving the right side also of the cranium. In both of Spiller's cases an endothelioma was found growing from the dura and pushing the brain before it. In both operation was attempted, but in the first case the bone of the cranium was so implicated by the tumor, and hemorrhage on this account so profuse, that all attempt to remove it had to be abandoned. The disappearance of nearly every symptom after opening of the skull in the second case was due to decompression. The cranium in Spiller's second case was very dense, but not infiltrated with tumor-tissue. Disappearance of all symptoms after removal of a brain tumor is exceedingly rare, but in this second case almost every symptom disappeared. The patient's speech is very nearly or fully normal, he has no hemiplegia, no optic neuritis, little or no paresthesia of the hand, no disturbance of writing, little or no abnormality of gait, and no exaggeration of the patellar reflexes, all of which things were present before the operation.

The presence of tumor now in 5 cases of hemicraniosis with operation or necropsy, and probably also in the clinical case of Brissaud and Lereboullet, should make one fear that any pronounced local thickening of the cranium is likely to be associated with a tumor growing from the dura, and operation should be attempted as soon as cerebral symptoms become manifest. It may be possible to remove the tumor in this way while it is yet small. Fortunately, this local thickening of bone is usually over or near the motor area of the brain, so that the symptom-complex is distinct, and the tumor growing from the dura is not likely

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(1) See Practical Medicine Series, 1903, Vol. x, p. 210, where Brissaud's original paper is reviewed.

(2) Jour. Am. Med. Assoc., Dec. 21, 1907.

to infiltrate the brain. Operations on these dural tumors are especially serious because of the severe hemorrhage, and they might be more successful if done in 2 stages.

(b) *Epicerebral Sarcoma.* A case of epicerebral sarcoma in the left sensory-motor area, removed by operation, and terminating in recovery, is reported by E. Tschudy and O. Veraguth.[1] The patient, 60 years of age, was well until her fifty-fifth year, when one morning she was awakened by jerking in her right foot, lasting 5 minutes. Later in the same morning the whole left side began to jerk, with the possible exception of the face. After this second attack she was unconscious for 3 hours. The attacks disappeared for a few months and then reappeared, the initial jerking always being in her right great toe, then in the foot and leg, then in the right hand and arm. These attacks of Jacksonian epilepsy lasted about 3 years, when she consulted one of the authors of this report, who advised immediate operation. She tried quack salves for a year, however, before she consented. In the meanwhile the left arm and leg had become paretic. There was no headache, dizziness, nor vomiting. The retinæ showed no choked disks or optic atrophy. Slight sensory disturbances were found in the right foot and hand, and she could not recognize objects in the right hand by her sense of touch. Babinski's toe-sign was present in the right side, the abdominal reflex was absent. In spite of the partially negative findings as to tumor, an operation was determined and performed in 2 stages, the blood-pressure falling to 60 mm. during the first step in the operation, thus compelling a two-step operation. At the second operation there was found beneath the dura a kind of membrane, apparently of deep-red, granulation tissues. It was very friable, but easily separable from the dura in a flattened mass 12 to 13 cm. antero-posteriorly, and 8 to 9 cm. wide. Improvement followed its removal, both the astereognosis and apraxia disappearing. The salient points are the absence of the general signs of tumor, such as headache, vomiting and choked disk, and the practical conclusion that the two-step operation is to be adopted in cases of this sort.

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(1) Corresp.-Blatt f. Schweizer Aertzte, June 1 and 8, 1908.

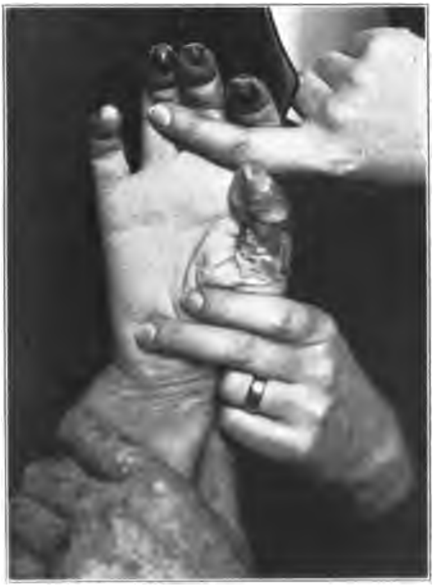

PLATE II.

The appearance of the thumb and finger tips on the twentieth day after operation.—W. W. Graves (page 115).

(c) *Neuroglioma Gangliocellulare.* T. M. T. McKenna and F. R. Proescher[1] report a case of neuroglioma gangliocellulare of the brain, with operation and recovery, in a male, 39 years of age, and a butcher by occupation. He showed the general signs of tumor, headache, projectile vomiting, and optic neuritis; and evidence that the neoplasm was in the right frontal lobe. McKenna says: "My reasons for this opinion were that the pain was most intense on the right side of the forehead, and he localized this pain with his hand, and there was the positive evidence of the emotional condition and the negative evidence of lack of any symptoms pointing to an involvement of the motor area of the parietal, occipital, or temporal lobes, or the base of the brain. (Furthermore, I had in remembrance a case of brain tumor where the patient had localized the pain by his hand. He had mental involvement alone, and I found on postmortem examination a tumor of the frontal lobe just underneath the place where he had localized his pain.)"

Operation was performed and a tumor the size of a pecan was shelled out from the second right frontal gyrus about 1½ inches from the median line. The operation was successful, and within 10 months the patient was wholly recovered. The tumor proved to be a neuroglioma gangliocellulare.

Operative Treatment. W. W. Graves[2] reports a case of brain tumor which was operated upon, the end result being complete recovery. The patient's illness began with motor weakness of the right extremities, paresthesia in the right forearm and hand, and a peculiar speech defect. In a few weeks she was seized with a convulsion of the Jacksonian type, beginning in the right hand, spreading to the whole body, and attended with loss of consciousness. When the latter was regained paralysis was found to be complete on the right side, and speech was lost. The ophthalmoscope showed "well nourished disks." The right forearm, hand, and fingers were distinctly cyanotic, and particularly the finger nails, which also showed distinct longitudinal corrugations. There was a slight right-sided

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(1) New York Med. Jour., July 18, 1908.
(2) Med. Record, May 23, 1908.

foot drop, without disturbance of motor tone. The Babinski, Oppenheim and Gordon reflexes were present on the right side, absent on the left. There were no sensory disturbances. The percussion note over the left superior fronto-parietal region of the skull was distinctly of lower pitch than the corresponding area on the right side. The patient recognized various objects and their uses, but could not name them, speech being reduced to "I can't" and "yes, yes."

She made rapid improvement despite 2 more convulsions, and regained her speech in about 10 days. A second ophthalmoscopic examination showed the nerve-heads to be clean cut, of a healthy pink throughout, and the retinal vessels were normal. Headache and nausea were absent. A preliminary diagnosis of "exhaustion paralysis" following a Jacksonian fit was made, the lesion being placed in left ascending frontal and parietal gyri, the arm center being the storm center. Since careful questioning disclosed symptoms dating back 3 years, a neoplasm irritative rather than destructive, was thought to be the cause, possibly a cavernous angioma or an arachnoidal cyst.

Operation was performed, and a thin-walled cyst found through which the greatly depressed gyri could be seen, the larger end overlying the ascending frontal, and the other the first and second frontal gyri. The postoperative phenomena consisted of a complete motor aphasia and right hemiplegia, of Jacksonian fits starting 6 hours after the operation, beginning in the right thumb and index finger, and rapidly spreading to the muscles of the upper extremity, shoulder, and face. In 4 days the fits ceased. Speech returned within 24 hours after the operation. It was noted at this time that every trace of rigidity had disappeared, that the Babinski and Oppenheim reflexes were absent, though Gordon's reflex was still present.

"During the first 24 hours after the operation the patient complained of intense pain in the right upper extremity, especially about the finger tips and nails, and particularly the thumb. On examining the extremity, the previously existing hyperemia had almost disappeared and the finger nails were paler than they had ever been before. A large bleb or blister formation was found involving the radial, palmar, and tip turfaces of right thumb. This increased from day to day and eventually involved fully one-half of the surface of the distal phalanx. Within the next 7 days, similar blebs appeared about the tips of index, middle, and ring fingers in the order named. For some hours preceding the formation of each blister, the patient complained of excruciating pain in tip of finger next to be invaded, and after the formation of a blister the pain became less. When the blisters first appeared they were translucent, but as time went on they became a brownish-black color, hard and dry, and were cast off in the next 3 or 4 weeks. The finger tips even beneath the sloughs, remained exquisitely tender and painful for some time. The patient continued to complain of unnatural sensations in the hand and fingers for several months following the operation." (See Plate II.)

Physical examination made about a year after the operation showed the patient to be well nourished, contented, happy, and in every way normal as to her nervous phenomena. The skin of the right hand and forearm was not cyanotic, and the finger-tips no longer hypersensitive.

Gummata of the Caudate Nuclei. Campbell[1] reports a case of gummata of both caudate nuclei, the function of which is vaguely known. They are doubtless thermic centers, and possibly have some motor function, although injuries of these nuclei result in only transitory paralysis unless the internal capsule is injured. A case of bilateral involvement of these nuclei has been reported by Hutchinson. The patient's first symptom was incontinence of urine and paresis of the sphincter ani. He was indolent, dull, stupid, and phlegmatic. There was no paralysis, but the gait was reeling. He emaciated rapidly and died. Hutchinson expresses his surprise that lesions so located should cause so much mental impairment without paralysis. Other isolated cases have been reported by Gowers, Charcot, and others.

The present case involved a young man of 22 years, who in October, 1906, was seized with a mental trouble making him unfit for work. On his arrival at the hospital the hands and feet were found to be strikingly cool and cyan-

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(1) Berl. klin. Woch., March 2, 1908.

otic, and the pulse small, ranging from 50 to 90 beats per minute. The temperature was subnormal, ranging from 95° to 96.8° F. His glance was dull and his face expressionless. Saliva trickled from the half-opened mouth. There were involuntaries both on the part of bladder and bowels. He had to be fed, because he either would not eat of his own initiative, or he would put anything into his mouth without regard to its nature. Left to himself he remained quietly in bed, hardly said a word spontaneously, and manifested no interest in his surroundings. He never showed evidences either of depression or of pleasure, but occasionally he laughed stupidly or made up a face of disgust. He never showed delusions or evidences of hallucinations.

Very striking were the motor symptoms. He showed a minimum of spontaneous motions. The tonus of the entire musculature was reduced; and strong reactions never occurred even after severe irritation. He reeled badly on attempting to walk, sometimes to the right, sometimes to the left. Later on in his illness he remained wholly in bed in a very peculiar position. Lying on his back he held his head flexed on his chest, and his thighs drawn up to the body. This posture could be easily changed passively. Shortly before death there was opisthotonos and stiffness of the neck muscles.

At the beginning of the sickness there was an evident paresis of the right lower facial trunk (cervico-facial division of the seventh nerve). The right leg and the right arm were not moved, and the power of the right side was somewhat less than that of the left. Yet there was no paralysis proper at any time. The reflexes were unchanged and the toe-sign of Babinski was absent. Sensitiveness to pain was everywhere present, but sensitiveness to touch could not be tested because of his dementia. Use of the right arm was clumsy, and often purposeless movements were indulged in, suggesting apraxia. There was very marked agraphia, both from dictation and copying. Speech was understood and simple commands obeyed. He carefully avoided all complicated movements, such as pulling on his trousers. Indeed he could not get out of bed later in his illness. In talking he moved lips and tongue very slowly, spoke monotonously, and permitted the saliva to run out of his mouth while trying to talk. He could read many words, others he got wrong, and when asked to name objects he succeeded sometimes and failed at others.

The slight right-sided hemiparesis and the disturbances of speech and writing aroused the suspicion of a left-sided focus. Nothing could be determined as to the nature of the focus. There were no evidences of tumor except an occasional bradycardia; and the retinæ were unchanged. There was no vomiting. No sign of syphilis could be discovered.

In the last few days of his life a paralysis of the left oculo-motor nerve developed, together with a left-sided facial paralysis. The left pupil was dilated and did not react to light. Shortly before death the right oculo-motor nerve began to be paretic.

The postmortem examination showed gummata in the kidneys, an old syphilitic scar in the trachea, and gummatous disease of both caudate nuclei, completely destroying their heads. The tails of the caudate nuclei were free on both sides. Meninges and cortex showed nothing. The paresis of the third pair of nerves proved to be due to a little gumma situated where they leave the midbrain. The right-sided paresis was transitory and does not therefore belong in the picture of caudate disease. The slight aphasia was found to be due to a gumma in the third left frontal convolution. There remain as symptoms proper to caudate nucleus disease, disturbances of temperature, psychic and motor disturbances, that is the disturbance of the position of the body and head, the disturbance of gait, the muscular atony, and the loss of spontaneous movements. The most certain of the cardinal symptoms is the temperature reduction, associated with bad heart-action, cyanosis of the extremities, and rapid loss of weight. The loss of spontaneity of movement indicates that the caudate nucleus has much to do with that function. Very prominent were the psychic symptoms, resembling apathetic dementia.

OPTIC THALAMUS.

Optic Thalamus Syndrome.

The optic thalamus syndrome as depicted by Déjérine was presented in the Practical Medicine Series, 1907, Vol. X, p. 119. P. Hartenberg[1] has written an excellent résumé of the syndrome. At present this is somewhat of a neuropathologic novelty, first described in 1903 by Déjérine and Egger, and amplified later by Thomas and Chiray. The latest comprehensive paper on the subject is that of Roussy, presented in his inaugural thesis.

Symptomatology. The essential symptoms are the following:

(1) *Hemianesthesia,* of organic type (in contradistinction to hemianesthesia of hysterical type), slightly developed for superficial sensibility (touch, pain, and temperature), but always very pronounced for deep sensibility (bones, tendons, muscles). Superficial sensibility is never completely abolished; it is only diminished with hypalgesia, paresthesia, delayed sensation; on the other hand, deep sensibility is profoundly disturbed. There is a considerable diminution of bony sensibility, a loss of the muscle-sense, abolition of all perception of active or passive movements, of the position of the limbs, of the sense of weights, and of resistance.

(2) *Intense pain* on the anesthetic side, in the face, in the forehead, cheek, orbits, in the limbs and joints, with superficial rather than deep localization. The pains are continuous, with paresthetic sensations of numbness, formication, tingling, etc. But under the influence of pressure, a pin-prick, of cold, or sometimes without any known cause, the pains become excessive and paroxysmal, being described by the patient as needle-thrusts, painful pinching, or stabs with a dagger. These pains are absolutely refractory to all analgesic measures of treatment.

(3) *Slight hemiplegia.* This manifests itself by slight facial asymmetry, hypotonus, and diminution of the muscular force, but without atrophy or contracture. This hemiplegia has a tendency to disappear from the start, and

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(1) **La Presse Méd., Jan. 15, 1908.**

may remain subsequently only in traces. The reflexes are a little exaggerated or normal. Babinski's toe-sign is variable and uncertain.

(4) *Slight hemiataxia* is present, being limited and restrained, accompanied by astereognosis, but never markedly compromising motor coördination or locomotion.

(5) *Choreo-athetoid movements* occur, consisting either of little choreiform movements of the extremities, or the slow and worm-like movements of athetosis.

Besides these essential symptoms which constitute the syndrome, the patients sometimes present others, such as disturbances of the sphincters, anal and vesical tenesmus, hemianopia, vasomotor, trophic and secretory disturbances. These are all accidental, and are due to neighborhood involvement instead of actual disease of the thalamus.

The onset in general is not by a stroke, but after some mental beclouding or a slight loss of consciousness lasting a few hours. The motor disturbances have a tendency to recede to a minimum, whereas the sensory symptoms and especially the pains tend to persist indefinitely in the same degree, or to augment in time, so that in the fully developed syndrome they much exceed their first intensity.

Pathologic Anatomy. The thalamic syndrome supervenes when the lesion destroys (1) the optic thalamus in the posterior third of its external nucleus, a part of the internal nucleus, the median portion, and the pulvinar, and (2) a small number of fibers of the posterior part of the internal capsule, next the optic thalamus. The functions of the parts destroyed easily account for the symptoms present. The subjective pains are due to the irritation of the central end of the conducting fibers interrupted by the lesion. The motor troubles, the hemichorea and hemathetosis, are due to the irritation of the neighboring capsular fibers. Hemianopia when present is due to involvement of the neighboring radiations of Gratiolet.

Diagnosis is difficult even for the experienced neurologist, because the thalamic syndrome can be duplicated by any lesion cutting the sensory pathway and just grazing the pyramidal tracts, whether in the region of the corpora quadrigemina, the peduncle, the pons or even the medulla. In all these situations, however, the diagnosis is apt to be settled by other accessory symptoms, on the part of the cranial nerve nuclei or other pathways, not present in the thalamic syndrome. For example, lesions of the corpora quadrigemina give a hemiparesis, a hemianesthesia for both superficial and deep sensations, hemiataxia, athetosis; also ocular paralysis in associated bilateral movements and diminution of hearing, the 2 latter symptoms not being those of thalamic disease. And in Weber's syndrome, though there is hemianesthesia, it is crossed, and there is also external strabismus due to paralysis of the third nerve, besides the hemiataxia, hemichorea, and hemitremor, common to this and thalamic disease. A lesion of the upper pons, besides causing hemiplegia, superficial and deep hemianesthesia, ataxia, and choreo-athetosis, produces also ocular paralysis of associated bilateral movements, with nystagmus on raising or lowering the eyes, dizziness, asynergy, and dysarthria. A lesion of the lower pons will cause besides the symptoms simulating the thalamic syndrome, paralysis of the facial and abducens nerve of the side opposite the lesion.

Since the thalamus contains no motor fibers, it doubtless would be better for this symptom-complex to be named the thalamo-capsular syndrome.

Clinical Case. Roque, Chalier, and Cordier[1] report a case in which they made a diagnosis of hemorrhage into the optic thalamus. The patient was a woman, 59 years of age, with a cancer of the liver, as determined by an exploratory laparotomy, and with the high tension pulse of a chronic nephritis, had some sort of a cerebral hemorrhage 12 days after the operation. On January 6, 1908, in the afternoon, there came on very gradually, without a stroke, a left hemiplegia, without evident participation of the face, with absolute loss of power. The patellar reflexes were normal and Babinski's toe-sign was absent. Plantar sensibility was preserved, with slight movements of retraction on stroking the sole of the foot. There was on the left side an almost complete loss of sensation to touch, pain, and temperature, except in the trunk and upper thigh. The deep sensibilities were blunted, and the muscle- and joint-sense was lost. After the onset of the hemi-

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(1) Lyon Méd., May 10, 1908.

plegia, there ensued severe pain in the paralyzed side, calling forth from the patient continuous complaint day and night. The sphincters were normal. There was complete insensibility of the cornea, conjunctiva, and lashes of the left eye, but in the right eye sensibility was intact. Hearing and smell were slightly dulled on the left side. Swallowing was not impaired. A month later the patient died, there having been no change in the symptoms, other than the development of delirium, and a sacral bedsore, with retention of urine and fever. The postmortem examination showed a hemorrhage which had almost completely destroyed the posterior half of the right optic thalamus. The cerebral peduncles, pons, cerebellum, and medulla were intact.

The diagnosis was difficult. At first it seemed unlikely to be an organic hemiplegia because of the integrity of the psychic state, and there was no disturbance of memory or of comprehension. Careful examination, however, showed disturbance of the deep sensibilities with loss of the muscular sense, except in the trunk and upper thigh. Hence hysteria was eliminated. The increased pulse-tension strongly suggested hemorrhage, but its site was problematical. A cortical lesion was not likely, first because the lesion was very likely a hemorrhage, and so not apt to be cortical; and also because the special senses were more or less involved.

In many recent publications Déjérine and his pupil Roussy have outlined the symptoms of disease of the optic thalamus and have reported 4 cases with postmortem examinations. The cardinal symptoms are (1) hemianesthesia involving all 3 sorts of sensation, and especially a deep hemianesthesia with loss of the muscular and stereognostic senses, and with hemiataxia; (2) pain, severe, persistent, sometimes intolerable, on the same side; (3) a slight hemiplegia, rapidly and more or less completely regressive; (4) possibly choreo-athetoid movements and hemianopia. In the above case the hemiplegia was more pronounced than in Roussy's cases, but had begun to disappear within a month, when death intervened. There were, therefore, no choreo-athetoid movements, for the same reason of early death, and no hemiataxia. On the other hand, 2 cardinal symptoms of the thalamic syndrome were clearly present: the hemianesthesia of an organic character, with special alteration of the deep sensibility, and the very great pain on the same side, so severe as to make the patient cry out continually, so that isolation was necessary in the interest of the other patients in the hospital.

MIDBRAIN.

Two very cleverly diagnosticated cases of **FOCAL DISEASE IN THE MIDBRAIN**, in the region of the corpora quadrigemina are reported by M. Infeld.[1] The first case was that of a man, 70 years of age, whose trouble developed after a fall when he was 5 years of age, and who spent the last 18 years of his life in a charitable institution. His objective findings can be divided into 2 groups: 1. Paralysis of the ocular muscles of the *left* eye rather extensively developed; the left eyeball was turned far over toward the outer canthus, and could be turned inward only a little, though it could be turned further outward; no other movement could be executed. The *right* eyeball was turned slightly out; abduction was almost completely possible, adduction about half-way possible, elevation and depression reduced to a minimum, with twitchings. The left pupil was wider than the right, but neither of the pupils reacted. Thus in addition to a bilateral paralysis of the trochlear nerves there was also on the left side a pronounced paralysis of the oculomotor nerve. 2. The remaining symptoms had to do with the right side of the body. The muscular and bony development of the right extremities took place properly, and they showed a very considerable degree of spastic paresis, combined with hyperflexibility of the fingers and disturbances of sensation. The deep reflexes of the lower extremity were clonic. The abdominal reflex was absent on the right side. There was no Babinski toe-sign. The whole side showed involuntary movements of a choreic type, and these were increased by voluntary movements just as an intention-tremor is so increased.

A *diagnosis* was made of *concretion in the left red nucleus* of the midbrain, and confirmed by postmortem exam-

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(1) **Wien. med. Woch., Aug. 17, 1907.**

ination and by further microscopic work done in Obersteiner's Institute. The localization was made by reason of the presence of Benedict's syndrome:—almost complete involvement of the nerves of one eye, except the abducens, and involvement of the trochlear nerve in the other. This placed the lesion in the tract leading from the nucleus dentatus of the cerebellum through the superior cerebellar peduncle to the opposite nucleus ruber in the midbrain. This pathway crosses the oculomotor pathway in the red nucleus, and so located the lesion. It was further localized by the contralateral hemichorea. The diagnosis of a concretion was based upon the fact that almost all similar cases proved to be due to tubercles appearing in childhood, notably in a case reported by Halban and the author from Obersteiner's Institute in 1902. The fact that the lesion in this case dated from the fifth year made this diagnosis almost certain. It is noteworthy that in this case the pes pedunculi and the pyramidal tracts were found intact.

The second case occurred in a woman, 25 years of age, with middle ear disease. The right ear showed signs of a healed up purulent otitis media, while the left ear showed a chronic, fetid, purulent discharge. The patient had been operated upon the day before. The lungs showed dulness and râles in the left apex, and diminished excursion of the right lower border. The patient was somewhat stuporous, but still was in a condition to be examined. Ophthalmoscopic examination showed a double choked disk. There was rigidity of the neck and sensitiveness to pressure, the vasomotor excitability of the skin was quickened, Kernig's sign was present, the abdomen was retracted, and there was a history of hyperesthesia, especially in the lower half of the body, for 2 weeks. Besides these plainly meningitic symptoms, there was a suggestion of a right ptosis and a right-sided slight facial paresis. The right pupil was wider than the left, slightly mydriatic, and both pupils were completely rigid to light, though very slightly responsive to accommodation. The day before there was very evident conjugate deviation of the bulbs. All of these eye-symptoms had existed for several days. Right hemianopia had also been observed previously, but was now absent. The power of the upper extremities was preserved, but their movements were uncertain, and had been previously very ataxic. The plantar reflexes were present and there was no Babinski toe-sign. For 2 years there had been headache, especially occipital, vertigo, and vomiting for 1 year. There was no suggestion of syphilis.

Besides a tuberculous meningitis, the existence of a tubercle was diagnosticated. The right ptosis and the dilatation of the right pupil together with the loss of the light-reflex in both pupils, indicated a lesion in the oculomotor nucleus. The transitory hemianopia indicated a lesion close to the optic tract; the transitory deviation indicated a lesion near the posterior longitudinal bundle. This placed the lesion beneath the right pair of corpora quadrigemina. The ataxia was ascribed to this lesion, while the facial paresis was ascribed to the meningitis. Since there was no paresis or hemichorea the pes pedunculi and the fillet were thought to be intact, and were found so postmortem. This autopsy disclosed a tubercle about the size of a hazelnut, which had pushed the right side of the midbrain toward the left, and had flattened the right pair of the corpora quadrigemina. A second tubercle was found on the dorsal side of the left cerebellar hemisphere, but this had caused no symptoms (except possibly the occipital headache).

CEREBELLUM.

CEREBELLAR TUMORS.

Symptomatology. Alfred Gordon[1] reports 6 cases of cerebellar tumor, 4 of which came to autopsy, all of them showing a peculiar position of the head. [Attention was called to this characteristic symptom in the Practical Medicine Series, Vol. X, 1904, p. 161.—Ed.] One of Gordon's cases may serve as an example of all. In his first case the striking symptom was the unavoidable necessity for the patient to hold his face turned to the right side; since, whenever it was turned to the left, a most intense vertigo would appear. The autopsy showed a soft tumor in the left cerebellar hemisphere. The second case also showed intense vertigo on attempts to turn her head to the left,

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(1) Jour. Am. Med. Assoc., Aug. 8, 1908.

which ceased when she was permitted to hold her head forward and inclined to the right.

"The 4 cases that came to autopsy are conclusive as to the relation of the attitude of the head to cerebellar disease. In the 2 purely clinical cases the symptoms of the cerebellar tumors were so typical that there is no doubt as to the diagnosis. In all 6 of the cases the head assumed a certain persistent position.

"In 4 cases it was turned to the side opposite the lesion during the entire course. In 2 of them it was inclined at first toward the seat of the lesion, but sometime before the patients died the head changed its position.

"Special emphasis is needed in regard to the increase of vertigo and headache when the inclined heads were turned forcibly toward the side of the lesion. This observation is quite novel. In the literature at my disposal I have been able to find one similar but not identical case published by Laruelle. He localized the tumor in the left cerebellar hemisphere because the head was inclined toward the same side. The tumor, however, was found on the right. The record also shows that each attempt to turn the head to the opposite side provoked an unusual pain."

E. Siemerling[1] reports 7 cases of tumor of the cerebellum very fully and ably, and upon them bases a paper upon the symptoms and treatment of cerebellar tumor. The characteristic symptoms of cerebellar tumor, according to the German school are 4: (1) cerebellar ataxia; (2) vertigo, continuous or paroxysmal; (3) nystagmus, with changes in the position of the ocular axis and perhaps certain paralyses of ocular muscles; and (4) ataxia, and in certain circumstances, tremor-like muscular movements in the arm or leg, especially evident in unilateral cerebellar disease on the side affected, and more so in the arm than in the leg. The generalized placcid paralysis or hypotonus on the side of the lesion neither Oppenheim or Bruns has ever seen.

Besides the 4 cardinal symptoms, there are numerous other neighborhood symptoms, which of course are not, and never can be, constant. The diagnosis can be made only by taking into consideration the whole course of the disease, the succession of the symptoms and their grouping. Among the symptoms is one emphasized by Oppenheim, namely the *absence of the corneal reflex,* and another discovered by Babinski, which he named diadokokinesia.[1] Oppenheim looks upon the reflex anesthesia of the cornea as the first sign of beginning trigeminal anesthesia, and as the indication of a tumor on the posterior forsa when the general signs of tumor are present. Oppenheim of course insists rightly that such reflex anesthesia must be unilateral to be of any value. In Siemerling's 7 cases corneal anesthesia was found in only 2, and in 1 of these the reflex at first was present. In all cases reported the corneal anesthesia has always been on the side of the tumor causing it. It is therefore extraordinarily valuable for deciding on which side the tumor is located.

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(1) Berl. klin. Woch., March 30 and April 6, 1908.

The cerebellar ataxia was variable. Very pronounced in cases 1, 2, 3, and 6; in 2 cases, 5 and 7, it was but faintly present, though in these cases a pronounced vertigo made up for its absence. In 1 case the vertigo was so great that the patient could not leave the horizontal position. In 4 other cases it was very marked, and it was present to a lesser degree in the 2 remaining cases. Oppenheim's contention that patients do not *fall* to the side on which the tumor is located is substantiated by 6 of these 7 cases. In case 3 only, did the patient pull to the side of the tumor.

Nystagmus was present 4 times, and absent 3 times. This is probably caused by compression of the corpora quadrigemina and of the pons, but it may be due to lesions of the cerebellum itself. A difference in the nystagmus as to whether the patient looked to the sound side or the side of the tumor was never found, but it may occur. An important rôle in the symptomatology of cerebellar tumors is played by the ataxic, tremor-like movements of the extremities. These movements most resemble the intention tremor of multiple sclerosis. They are important in indicating the hemisphere affected. In only 2 cases were they absent; in the other 5 cases they constituted an important symptom. As a rule the movements occur in the arm on the side of the lesion. Babinski's diadokokinesia was present once. Psychic disturbances are very rare, but halluci-

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(1) See Practical Medicine Series, 1903, Vol. x, p. 162.

nations were present in case 7. Lumbar puncture is important, especially for ruling out hydrocephalus internus. If the cerebrospinal fluid is removed slowly, and the pressure not allowed to fall below 100 mm., there is no danger from lumbar puncture. Frontal headache was intense in 3 cases.

As to treatment, palliative trepanning is advisable, especially when optic atrophy threatens; and if the tumor can be accurately localized, operation may be proposed.

Special Tumors. (a) *Ependymglioma.* M. Martens and W. Seiffer[1] report a case of cerebellar tumor in a boy complaining of headache, vomiting, and diplopia. There was vertigo, but no disturbance of gait. There was nystagmus. The corneal reflex was not affected on either side. There was no ankle-clonus or Babinski toe-sign. There was neither ataxia nor loss of power in all 4 extremities. No diadokokinesia was present. The left knee-jerk was weaker than the right. Percussion of the head in the left occipital region caused pain. Sensation was intact. With closed eyes there was a tendency to fall to the right. The diagnosis of a cerebellar tumor in the left hemisphere was made and operation determined upon. This was not successful, the patient dying shortly after it was performed.

The postmortem showed an ependymglioma which practically filled the fourth ventricle, growing from the ependyma forming its roof. In size it equaled a walnut. It was soft and white to rose-red. It completely occluded the foramen of Magendie. It exerted pressure on the pons, medulla and vermis cerebelli only. On opening the cerebral hemispheres similar tumors were found in the other ventricles. In the left posterior horn there was a tumor; and another in the left inferior horn. Another, the size of a cherry pit, was found in the bottom of the widely dilated third ventricle.

(b) *Cerebellar Tumor in Recklinghausen's Disease.* Courmont and Cade[2] report a case of Recklinghausen's disease with tumor of the cerebellum. The coexistence of this disease and symptoms of brain tumor has already been reported, but only very exceptionally. The authors show that the cerebellar tumor observed in this case was the

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(1) Berl. klin. Woch., Aug. 10, 1908.
(2) Lyon Méd., July 5, 1908.

result of the generalization of the neuro-dermo-fibromatosis of Recklinghausen. The patient, a female, 28 years of age, complained of pain in the head and trouble in walking. Since her eleventh year she had had in the left submaxillary region a tumor which had progressivly augmented. It was not painful and did not much disturb the patient. For some time it had remained stationary in size. In January, 1907, headache began, at first moderate, later much more severe. Then her gait became affected, so that by October, 1907, she had ceased to walk at all. She entered the hospital in March, 1908, with a continuous headache of varying intensity, but almost exclusively occipital and bilateral. Moreover she had had several attacks of vomiting of the cerebral type.

Examination showed that it was impossible for her either to stand or walk, except with support; and if supported, the gait was cerebellar in type. Muscular force remained intact. The reflexes were everywhere exaggerated, and briefly at times, patellar and ankle-clonus were present. There was no Babinski toe-sign, sensory disturbances or sphincteric involvement. Vision was diminished, but hearing and smell were intact. Beneath the left side of the inferior maxilla was a large soft, pseudo-fluctuating tumor. On each side of the neck and on the trunk there were a number of small cutaneous tumors, some sessile, some pedunculated, soft in consistency. These tumors coexist with pigmented patches of various sizes and tints. Visual acuity was reduced one-third, there was double choked disk, and evidence of a recent retinal hemorrhage in the left eye. Nystagmus was present.

The symptoms of Recklinghausen's disease are clearly complete: a large or major tumor in the left submaxillary region, subcutaneous minor tumors in the neck and trunk; and lastly pigmented patches. Asthenia and psychic disturbances were not appreciable. Whether the tumors were congenital cannot be affirmed with certainty, the first history of them dating back to the patient's eleventh year. The remainder of the symptoms point to a cerebellar tumor, especially the occipital headache, the severe disturbances of equilibrium with preservation of muscular strength, the exaggeration of tendon-reflexes, the absence of localizing signs such as aphasia, paralysis, and Jacksonian epilepsy, the rapidity of development and the intensity of the symptoms. The escape of the auditory, facial and oculomotor nerves suggested an intracerebellar tumor, and the tendency to fall backwards pointed to the worm. Because of the neurofibromatosis, the tumor was thought to be a neurofibroma.

After her entrance she began having epileptiform crises, with obnubilation, although without complete loss of consciousness. There were some movements of the forearms and a convulsive deviation of the globes of the eyes. Vasomotor disturbances, such as blushing, and abundant perspiration accompanied the attacks. In some of the crises there was incontinence of urine. Moreover there were respiratory difficulties and tachycardia, indicating compression of the floor of the fourth ventricle. Sudden death, common in cerebellar tumors, was feared, and occurred April 7.

At the autopsy, on opening the dura, a large amount of cerebrospinal fluid poured out. The ventricles were dilated. The right lobe of the cerebellum bulged and its surface appearing pale and compressed. Within was the tumor which had invaded the worm. It was firm, white, and merged insensibly into the surrounding nerve-tissue. The cervical tumor was found to be a neurolipoma. The cutaneous tumors were dermofibromata. Section of a pigmented area showed pigmented masses in the deeper layers of the epidermis. The cerebellar neoplasm was a neurofibroma which had undergone malignant degeneration.

TUMORS OF THE CEREBELLO-PONTILE ANGLE.

The *diagnosis* of tumors and other lesions in the cerebello-pontile angle is discussed by T. H. Weisenburg,(1) his paper being "concerned with the discussion of the value of different symptoms found in such lesions, and the report of 5 cases, in 2 of which a tumor growing from the dura covering the petrous portion of the temporal and the occipital bones gave some of the symptoms of a lesion in the cerebello-pontile angle, to such an extent that an operation was performed in 1. In the third case, there was entire absence of headache, nausea, vomiting, vertigo and choked disc, and yet sufficient symptoms were present to indicate a lesion in the cerebello-pontile angle, to find at operation a cyst, with the consequent, almost total, recovery of the patient; the report of a case of abscess of the pia which simulated the symptoms of a tumor in this angle, and, lastly, the report of a case in which a tumor of the fourth ventricle, compressing the inferior vermis, indicated a lesion in the cerebello-pontile angle. These cases are selected for report among others because of their rarity, and as illustrations of the difficulty encountered in diagnosis. In 3 of these cases a necropsy was done."

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(1) Jour. Am. Med. Assoc., April 18, 1908.

Tumors of the cerebello-pontile angle are as a rule fibromata, and grow principally from the eighth nerve, less frequently from the fifth and seventh nerves. They may spring from the cerebellum or pons, in which case they are not fibromata; and thirdly, they may grow either from the dura over the petron or occipital bone or both.

Symptoms. If a tumor grows from the eighth nerve there is at first diminution of hearing on the same side, with such irritating phenomena as buzzing, rushing, ringing noises, etc. Later deafness follows. Next incoördination results from pressure on the lateral lobe of the cerebellum, and the general signs of tumor appear. If a tumor grows from the fifth nerve the first symptoms are numbness and pain in the face on the same side, followed by anesthesia, and motor paresis if the motor root is involved. If a tumor grows from the seventh nerve, it may cause fibrillary twitchings in the facial territory and sometimes convulsions, Jacksonian in character, as in Weisenburg's second case. It is important to recognize these, for such Jacksonian convulsions are usually ascribed to the cortex.

"If a tumor grows in the cerebellum and secondarily involves the structures in the cerebello-pontile angle, the first symptom will, of course, be those resulting from cerebellar disease, and further symptoms will depend on what cranial nerves are involved. Occasionally, however, a tumor growing from the choroid plexus in the fourth ventricle, and compressing the middle lobe of the cerebellum, may give rise to the symptoms of a cerebello-pontile lesion, as in Case 5, in which the tumor grew outward from the fourth ventricle, into and involving the sixth and seventh nerves in the cerebello-pontile area.

"If a tumor grows from the pons, and secondarily involves the structures in the angle, the early symptoms will depend on whether the motor or sensory fibers are involved, this causing spastic paresis or disturbance of sensation on the opposite side of the body.

"As in 2 of the cases reported here, if a tumor grows from the temporal or occipital bones, and then involves the structures in the cerebello-pontile angle, the early symptoms may not be manifested until pressure is exerted on the different cranial nerves, pons or cerebellum. In the 2 cases reported here, the first symptom in Case 1 was right hemiparesis, followed by headache, increasing dizziness, cerebellar gait, disturbance of vision, left external rectus palsy, irritating noises in the ear, later complete nerve deafness, difficulty in eating, talking, and swallowing, vertigo, nystagmus, weakness of the left seventh nerve, and later on involvement of the right palate and right vocal cords, ataxia of all the limbs, especially on the left side, a spastic condition on both sides, more on the right, with increased reflexes.

"At the necropsy, a tumor was found, which grew from the dura of the petrous portion of the left temporal and occipital bones, and secondarily involved the cerebello-pontile angle. Evidently, in this case, the early symptoms were those resulting from general intra-cranial pressure, and secondly of an involvement of the cerebellum and nerves in the angle and those which resulted from the compression of the anterior portion of the pons and, lastly, some of the cranial nerves on the other side. See Plate III.

"The symptoms of lesions growing from the dura covering the petrous portion of the temporal and occipital bones and involving the cerebello-pontile angle secondarily will be bilateral, the greater involvement being on the side of the tumor. There should be present implication of the cranial nerves on both sides, with difficulty in eating, talking and swallowing, because of the involvement of the bulbar nerves, incoördination in the limbs, depending on the side of the cerebellum diseased, the general symptoms of brain tumor, and, at times, spastic paresis of the opposite side of the body because of pressure on the pons, all of these coming on gradually.

"Occasionally cysts will grow in the cerebello-pontile angle, but their diagnosis will offer no special difficulty, for the symptoms will depend on the cranial nerves involved and the extent of implication of the cerebellum and pons. It should be remembered, however, that cysts may not cause as much pressure as tumors, and, therefore, the general symptoms of intracranial pressure may be absent, as in Case 3, in which there was no headache, nausea, vomiting, vertigo or even choked disc, and the diagnosis was made on the involvement of the left sixth, seventh and eighth nerves and the left cerebellar symptoms.

"Rarely, as in Case 4, will an abscess in the pia give the symptoms of tumors in the angle. The diagnosis should not, however, offer very much difficulty for, as in this case, there will be a history of a middle ear disease, with secondary meningeal involvement, and there may be paralysis of the sixth, seventh and eighth nerves and some cerebellar symptoms. It is interesting to note that so far as known by the writer this is the only case of pial abscess correctly diagnosticated in life and proved by microscopical examination."

General symptoms of tumors of this angle. Headache as a rule comes on early, is mostly occipital, and is apt to be limited to the side of the lesion. Nausea and vomiting were absent in 3 of the 5 cases. Vertigo is one of the commonest and earliest symptoms, being described as giddiness, dizziness, a fainting feeling or a sense of rotation, either of self or of external objects. Choked disc or optic neuritis was present in 4 of the 5 cases, and is as a rule more marked on the side of the lesion. Excluding the symptoms resulting from direct involvement of the cerebellum, the diagnosis of tumor of the cerebello-pontile angle is based upon, first, the general signs of tumor, and secondly, upon the implication of the cranial nerves situated in the cerebello-pontile angle. Unilateral paralysis of even the ninth, tenth, eleventh and twelfth nerves sometimes results from pressure, so that there may be difficulty in eating, talking and swallowing.

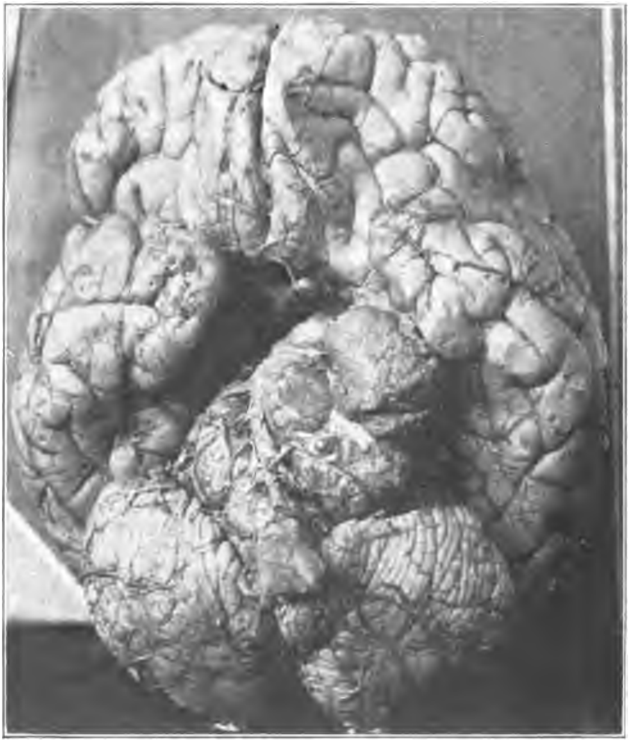

PLATE III.

Sarcoma growing from the left temporal bone, invading the left cerebello-pontile angle, giving symptoms difficult to differentiate from those of cerebral syphilis.—T. H. Weisenburg (page 129).

The cerebellar symptoms consist of cerebellar ataxia, shown in the gait, asthenia and atony of the muscles of the limbs and trunk, incoördination of the movements of the eyeballs If the tumor compresses the foramen of Magendie there will be hydrocephalus internus.

CEREBELLAR ABSCESS.

W. Trotter[1] reports 2 cases of cerebellar abscess and enumerates the common *symptoms* of the affection. "Cerebellar abscess produces or may produce (1) general evidences of an infective process, (2) evidences of a lesion in the brain substance spreading with variable rapidity, but as a rule subacutely, (3) evidences of a lesion of the lateral lobe of the cerebellum.

"1. General Evidences of an Infective Process. When the case is seen from the beginning there will practically always be a period of onset during which the symptoms are those of an acute infective process—a rigor, high fever, headache, loss of appetite, wasting." For several days there may be no evidence of brain involvement, signs of septicemia alone being present. Sometimes the abscess is latent, but rarely so in cerebellar abscess.

2. The general cerebral signs are headache, vomiting and optic neuritis, and these may be passed over as being well known. Optic neuritis is, however, often absent in cerebellar abscess. The headache is most commonly occipital and practically never lateral. As to the matter of consciousness, all stages of impairment occur, from slight dullness and disappearance of spontaneity, to profound coma. Yet while the lethargy and indifference gradually deepen, as long as the patient can be made to respond it will be found that intelligence is quite normal. It is unusual for any true delirium or confusion to develop.

3. Localizing Cerebellar Signs. These are extrinsic and intrinsic. (a) *Extrinsic signs.* In the posterior cranial fossa the structures other than the cerebellum which may be involved are certain cranial nerves and the pons and bulb. Cranial nerves. "The last 6 of these are those which have a more or less considerable part of their course in the posterior fossa. It is clear that they fall naturally

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(1) Brit. Med. Jour., Mar. 14, 1908.

into 3 groups—an anterior or superior, including the fifth and sixth nerves; a middle group, including the seventh and eighth; and a posterior, including the ninth, tenth, eleventh, and twelfth. The posterior group is very rarely involved by abscess of the cerebellum in such a degree as to be of diagnostic value. Some degree of defect in articulation is, however, not uncommon, but naturally enough it is difficult to decide how much of this may not be due to the mental state. The second group is of practically no value in diagnosis, seeing that involvement of it is much more likely to be due to the middle-ear disease which has caused the cerebellar abscess than to the latter itself. We are left, therefore, with the anterior group.

"It might have been expected that affection of the fifth cranial nerve would not be uncommon in cerebellar abscess, yet as far as present experience goes it would appear that the fifth as a rule escapes. It is probable, however, with more exact methods of determining sensory disturbances of slight grade we may ultimately find that changes in the cutaneous area of the trigeminal are not uncommon in cerebellar abscess.

"The case is quite different with the sixth nerve. Paresis of the external rectus on the same side is the commonest isolated sign of cranial nerve involvement met with in cerebellar lesions. Unfortunately, the diagnostic value of weakness of the external rectus is but small. It occurs not uncommonly in acute mastoiditis, and even otitis media, and is, therefore, of much less significance than is a weakness of conjugate movements, in which are implicated the external rectus of the diseased side and the internal rectus of the other."

Pons and Medulla. "Pressure may cause a hemiplegia of the upper motor neurone type, usually implicating leg and arm only, and accompanied by exaggeration of deep reflexes, ankle clonus, and extensor plantar response. Such a hemiplegia is much less likely to occur in abscess than in tumor, and it implicates the limbs on the side opposite to the lesion, as the pressure is above the decussation of the pyramids.

"*Intrinsic Signs.* The fact of the cerebellum being an organ concerned with the regulation rather than with the origination of movement, gives to the signs of lesions of it a quality characteristically different from lesions of the direct motor path. The contrast is well brought out when we remember in cerebellar lesions the great rarity of convulsive attacks, the absence of rigidity, and the fact that paresis is a late sign of advanced disease and scarcely ever amounts to paralysis.

"The motor effects of a cerebellar abscess may be classified as follows:

"1. Ocular signs:
    Nystagmus.
    Weakness of conjugate movements.
    Skew deviation.
"2. Signs affecting the limbs:
    Incoördination.
    Paresis.
    Hypotonicity."

As to signs affecting the limbs it may be said that disturbances of coördination may be demonstrated in several ways:

(a) By pointing to a given object, or finger-tip-nose movement.

(b) By the gait; wide base of support, one-sided staggering, difficulty in turning.

(c) By rapidly reciprocating movements (diadokokinesis).

(d) By the execution of complex habitual movements involving several joints. "When a normal person lying down is asked to straighten the lower limb from a position of flexion of both knee and hip, the movement executed is single, the extension of the knee and hip occurring together. Sometimes in cerebellar lesions this synergic movement is interfered with, and the patient straightens the knee first and then the hip."

As to paresis, "it is on the side of the lesion, does not involve the face, is not accompanied by rigidity, tends to involve the larger movements rather than the finer and is not associated with ankle-clonus or extensor plantar response. It is, however, not enough to say that there is no rigidity; there is, in fact, very often a striking flaccidity of the paretic limbs, as can be shown by passive flexion of the hip with extended knee, or by the possibility of producing slight hyperextension of the knee (knee angle sign)."

"*Reflexes.* The deep reflexes are often affected by intrinsic cerebellar lesions in such a mixed way as to produce a picture almost characteristic in its irregularity, but very difficult to generalize. We may say in a broad way that any reflex may be exaggerated or reduced, and both changes may occur with different reflexes at the same time or with the same reflex at different times; and that, therefore, the localizing value of such changes is often slight as regards the side of the cerebellum involved. For example, the knee jerk on the side of the lesion is often exaggerated, but it may equally well be reduced. It is possible, however, to be more definite as to the absence as a sign of intrinsic cerebellar disease, of the Babinski toe phenomenon and its variants, the 'fan sign,' Schäfer's, Oppenheim's, and Remak's reflexes."

Treatment. Prompt operation is very urgent, because of the risk of sudden death from respiratory failure. The case, once the diagnosis is made, should be looked upon as quite as urgent as one of perforated gastric ulcer or rupture of the spleen. The danger bears no relation whatever to the severity or mildness of the symptoms and signs. The chance of failing to find a small and deep-seated cerebellar abscess is considerable, so that a considerable area of bone should be removed and the dura incised freely, so that if an abscess is present it can extend more freely toward the surface, and so prevent sudden pressure on the medulla, or at least minimize it.

E. B. Dench[1] reports a fatal case of cerebellar abscess in a boy of 13 years in whom there were latent symptoms of cerebellar tumor, until suddenly fever rose rapidly and dullness and stupor followed. The author analyzes 102 cases collected from the literature. The *route of infection* was through the petrous portion of the temporal bone in 30 cases, through the lateral sinus in 30 more; in 4 the infection seemed to be through the mastoid, in 3 the abscess was secondary to cerebral abscess, while in the remainder the route of infection was not given. It would

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(1) Am. Jour. of the Med. Sciences, Nov., 1907.

seem, therefore, that cerebellar abscess occurs with about equal frequency through the internal auditory meatus, and through the aqueductus cochleæ and aqueductus vestibuli. The most prominent and constant general *symptom* is headache, occurring in 71 of the cases. Next in frequency was vomiting, occurring in 54 cases, and vertigo was present in 30 of the 102 cases. Nystagmus occurred in 17 cases, bradycardia in 40 cases. In 26 cases the temperature was subnormal. Retraction of the head was noted in 12 cases, stupor in 44 cases, varying from lethargy to coma. Other occasional symptoms reported were increase of the knee-jerk on the affected side, absence of the knee-jerk, inequality of the pupils, dilatation of the pupil on the affected side, strabismus, Cheyne-Stokes breathing, and optic neuritis. The latter was present in 34 cases, absent in 37, and not reported in 31. Of the 102 patients only 33 recovered.

As to operative treatment, the method must depend upon the probable route of infection which is often difficult or impossible to determine. If this cannot be determined the surgeon should enter anterior to the lateral sinus.

FAMILIAL CEREBELLAR DEGENERATION.

Gordon Holmes[1] describes a form of familial degeneration of the cerebellum, occurring in 4 members of a family of 8, 1 of the 4 having been examined postmortem. The cases described are thought by him to have a bearing upon the disease described by Marie as hereditary cerebellar ataxia, made up of the cases of Fraser, Nonne, Brown, Klippel, and Durante, though the 4 cases here presented surely do not belong to that group. In each of these 4 cases the disease set in between 33 and 40 years of age and progressed slowly till death, which resulted from an intercurrent affection at an advanced age. "The first and most prominent symptom was a reeling or staggering gait, like that of a drunken person, evidently due to incoördination of the movements of the lower limbs or to defect in equilibration; the next symptom to appear in each case was irregularity in, or uncertainty of, the movements of the

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(1) Brain, Part iv, 1907.

upper limbs; and later articulation was affected, it became hesitating or scanning and explosive. Tremor of the head and limbs was mentioned in the notes of all the cases, and was referred to as a prominent symptom by the sister who was interviewed. Nystagmus, or more probably irregular nystagmoid jerkings of the eyes on movement, was observed in the 3 cases of which accurate notes are available. There was no defect of vision or of hearing, and no motor paralysis or sensory disturbance was detected. The tendon-reflexes were unaffected or slightly exaggerated, but ankle-clonus or other certain evidence of disease of the cortico-spinal tract was not obtained in any of the cases. The sphincter functions were intact, and there was no obvious mental impairment.

"The anatomical basis of these symptoms, which it may be assumed was the same in the first 3 cases as in Case 4, was a degenerative affection of the cerebellum. The disease was limited to this organ, as the inferior olives were the only other part of the nervous system in which pathological changes were found, and in these it was only secondary and of no significance in the production of the symptoms. The disease was a primary and progressive degeneration of the nervous elements of the cortex of the cerebellum and of the fibers which spring from them. The most prominent change in the cortex was the disappearance of the Purkinje cells, which was complete in the greater part of the cerebellum."

In a lengthy critique in the same number of *Brain,* Holmes thoroughly reviews the literature on Marie's Hereditary Cerebellar Ataxia, enumerating 66 articles in his bibliography, and concludes as follows: "Any justification there may have been for including the cases originally collated by Marie under the title 'Hereditary Cerebellar Ataxia,' has been shattered by the anatomical examination of the central nervous system." In some of these cases the cerebellum has been found postmortem to be normal, as in the cases of Klippel and Durante, and of Sanger Brown. The majority of cases reported since Marie's paper as cases of hereditary cerebellar ataxia seem to be merely olivo-ponto-cerebellar atrophies, and in not a single one of these were the symptoms either hereditary or familial. "We are consequently driven to the conclusion that no form of disease exists to which the term 'Hereditary Cerebellar Ataxia' can be aptly applied."

DISEASE OF THE PONS.

PONTILE HEMORRHAGE.

Theodore Diller reports a case of pontile hemorrhage with autopsy.[1] References to pontile hemorrhage are sufficiently rare to justify the report of the case. Guillian mentions among its *symptoms* vomiting, irregular respiration, polyuria, single or double hemiplegia, paralysis of the lower face and of the tongue and larynx. Erb names elevation of temperature as a significant sign. Dana, from a study of 190 cases of pontile disease, gives as the more frequent causes of pontile hemorrhage, nephritis and arteriosclerosis, chiefly of the transverse and median branches of the basilar artery. Dana's description of the symptomatology of pontile hemorrhage follows: "The patient has some prodromal headaches and malaise for a few days, with vertigo and sometimes vomiting. Then he falls suddenly, as if by a lightning stroke, into a coma, usually very profound. There are twitchings of the face or of the limbs, or both, but rarely any general convulsion (3 cases). The face is flushed, there is a fall of temperature, and the pulse is irregular, but not markedly increased or slowed; as a rule, the respiration is slow, 4 or 6 per minute, or more often irregular and of Cheyne-Stokes type. The pupils are contracted to a pin-point, and do not respond to light, but may be uneven. There is convergent strabismus or conjugate deviation of the eyes. The limbs are at first stiff, but may be relaxed later and the reflexes increased.

"The patient cannot be aroused, but can be made to vomit. A few hours later the temperature rises, sometimes very high—106° to 108°—but usually not. There are involuntary movements of the bowels, swallowing is impossible; evidences of dyspenea are shown. The pulse becomes more rapid, cyanosis develops, and the patient dies

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(1) **Am. Jour. of the Med. Sciences, March, 1908.**

in from 6 to 20 hours, usually with evidence of paralysis of respiration.

"If the hemorrhage is less severe, the coma is less profound or may be absent, the patient shows signs of agonizing suffering and difficulty in respiration, evidences of crossed paralysis, crossed anesthesia, and disturbance of equilibrium and ataxia can be noted."

Oddly enough, he makes no mention of double hemiplegia in this description. But in consideration of the structure of the pons, it will be readily seen that there may be either single or double hemiplegia, or crossed paralysis; or paralysis of the fifth, sixth, or seventh nerves may occur, depending upon the location and size of the hemorrhage.

The case reported by Diller occurred in a woman aged 63 years, long an inmate of an infirmary, to which she was admitted in 1871 on account of delirium tremens. During the past 15 or 20 years she showed a condition of secondary dementia. Her seizure began with an attack of vomiting and purging, with some clouding of consciousness, but no paralysis. Her pulse was full, strong, and compressible, and about normal in rate. On the morning of the next day she was unconscious. Her respirations were hurried and stertorous. Both arms upon examination seemed to be paralyzed, and a double hemiplegia was thought to exist. The pupils were small and immobile, but equal. The diagnosis of pontile hemorrhage was ventured on the basis of these points: double hemiplegia, myosis, and the suddenness of onset with vomiting. Death followed 30 hours later, no new features having developed.

The postmortem examination was made 20 hours after death. "The vessels at the base of the brain were discovered to be in a state of advanced degeneracy, being hard, tortuous, and calcified in many places. Over the entire area of the pons, beneath the pia mater and extending down over the oblongata for three-quarters of an inch, was a large, thick hemorrhage, firmly coagulated (see Plate IV). The exits of all the cranial nerves from the third to the eleventh were involved in the clot." The clot was about one-eighth of an inch thick. There was some hemorrhage under the pia and between the cerebellum and the

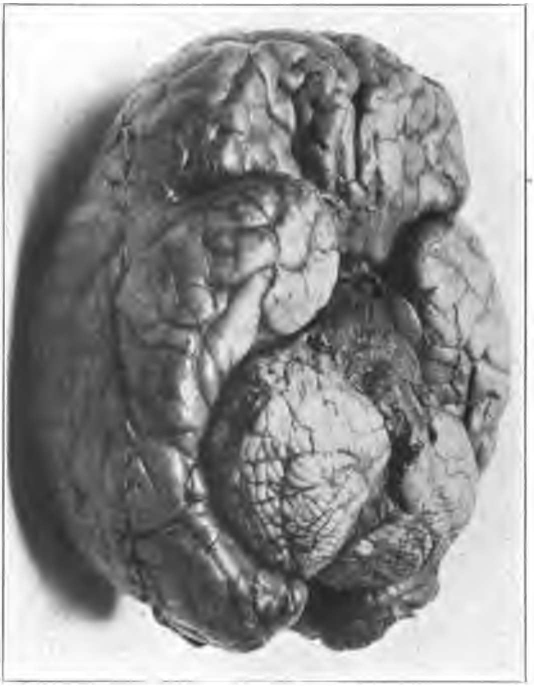

PLATE IV.

Photograph of a brain, showing hemorrhage over the entire pons and the upper medulla oblongata.—T. Diller (page 139).

cerebrum, which had probably come from the pontile hemorrhage. There was no hemorrhage into the interior of the pons.

MEDULLA OBLONGATA.

SYRINGOBULBIA.

Ludwig Euders[1] reports a case of syringobulbia with postmortem report. The patient's case was reported by H. F. Müller in the Deut. Archiv. of Clin. Med., Vol. 52, p. 259, several years ago. Since 1893 the patient has been a Diener in the medical clinic at Munich, and did his work satisfactorily, managed to walk without much disturbance of gait, had learned to avoid burning himself, but developed anesthesia in the region of the right trigeminus. He died of broken compensation of the heart. A very careful postmortem examination was made, the details of which are given. The case proved to be a typical lateral syringobulbia, such as has been thoroughly described by Schlesinger: lateral and asymmetrically situated gaps without connection with the central canal; the epithelium of the latter remaining uninjured, even where the gaps extend up very close to it. The cavity itself possesses no epithelial lining whatever. Its wall is made up of a gliomatous tissue, irregular, rarefied in some places and thickened in others. Typical were the disturbances of the nerve-tissue, and the thickening or hyaline degeneration of the vessel-walls, and the dilatation of the perivascular spaces. Especially noteworthy in this case is the fact that the cavity extends forward through the whole pons.[2] A large fissure was found starting in the closed medulla, passing obliquely to the right, and being confined to that side, involving the right facial nucleus, and reaching to the nucleus of the sixth cranial nerve. The involvement of the pons is recorded as unique [though Spiller's case in this country has the priority, notwithstanding the claim of Euders of priority for his case.—Ed.]. Very interesting is the further fact that the contralateral olive and accessory olivary nuclei were completely atrophied. In this case there was

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(1) Deut. Archiv. f. Klin. Med., July, 1908.

(2) See Practical Medicine Series, 1907, Vol. 8, p. 160, where Spiller's case is recorded.

undoubtedly a close etiological connection between the disease of the vessels and the syringobulbia. Clinically some of the symptoms began with apoplectiform onset and were increased in intensity by sudden exacerbations, thus strengthening the pathologic evidence of the vascular origin of the malady. The gliomatous growth in this case seems to be merely a secondary, reparative phenomenon rather than the primary cause of the disturbance.

O. P. Gerber[1] reports 2 cases of involvement of the medulla in syringomyelia, but neither of them is accompanied with postmortem findings. Both of them showed trigeminal symptoms, and both showed corneal anesthesia, a condition emphasized by Oppenheim in the diagnosis of tumors of the posterior fossa of the base of the skull.

DISEASES OF THE SPINAL CORD.

Spinal Cord Injury.

Injuries of the spinal cord are discussed by A. R. Allen,[2] and nine cases, the cords of which were all carefully examined in Spiller's laboratory, are reported. Six of these cord injuries followed fracture-dislocations; one was due to a gunshot wound; one was due to a self-reduced dislocation without fracture; and one followed spinal concussion without injury to the spinal column.

Treatment. "When are we to recommend operation in cases of fracture-dislocation of the spinal column? In answering this question we must not base our advice on the isolated cases reported in which brilliant results have come after radical operative procedure. These cases are often will-o'-the-wisps leading to disaster, because we are as yet too ignorant of the processes of regeneration and spinal surgery to be able to see what factor in a particular case made for success, and in our ignorance we are prone to ascribe it to superlative surgical technic, promptness in operating, etc., factors any one of which is easily within our power and which if they were the true crux of the question would forever set aside the dispute on the subject.

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(1) Wien. med. Woch., Aug. 24, 1907.
(2) Jour. Am. Med. Assoc., March 21, 1908.

"A compound fracture of the spinal column, whether caused by gunshot or other violence, is often a suitable case for operation. Here there is not the conversion of a simple into a compound fracture. Do as little as possible in the operation in the way of removal or reposition of fragments. Of course, should one find a lamina driven into the spinal cord, it would be indicated to remove it; but rather than clear the premises of all existing débris, trust to the natural processes of organization and ankylosis, with the aid of immobilization, to bind the fragments, which one might otherwise tediously dissect away, out of reach of the spinal cord. When the canal is opened the greatest care should be exercised in sponging so that the spinal cord is not forcibly pressed on. Clots of blood can be removed by a stream of warm sterile physiologic salt solution, after sufficient time has been given for the oozing in the small vessels to cease spontaneously."

In the discussion following the reading of the paper, W. G. Spiller complained that Dr. Allen had failed to give any conclusions. The subject of laminectomy, Spiller said, is one of great importance. After examining many cases of fracture of the vertebræ, clinically and pathologically, Spiller believes that in most cases an operation may do harm. There may be exceptions, but in the majority of cases the patient is better treated by other means than surgical. Dr. H. M. Thomas also agreed that operation in cases of this character has been of no benefit.

The reflexes in a case of crushing wound of the spinal cord. A. Pitres[1] reports a case in which the spinal cord was crushed by a fracture-dislocation resulting from a boy's fall from a cherry-tree; and he discusses the condition of the reflexes and of the deep visceral sensations associated with the paraplegia. The present belief is that the centers of reflex activity are in the upper levels of the cerebrospinal axis, and that the stimuli producing the reflex act ascend through a great part of this axis; hence complete section of the cord in the cervical or dorsal region is followed by total loss of muscle-tonus, and of the cutaneous and tendon-reflexes below the point of injury. In the present case paraplegia immediately followed the fall,

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(1) Jour. de Méd. de Bordeaux, Oct. 13 and 27, 1907.

the wound being in the middorsal region. Cutaneous sensibility was totally abolished below the sixth or seventh dorsal vertebra.

The knee- and Achillis-jerks were totally abolished, even on reinforcement. The abdominal, cremasteric and plantar reflexes were also entirely absent. Strong irritation of the sole of the foot produced a small motor response, quite unlike the usual one in that it was very slow and deliberate and a full half second late. The movement produced was one of flexion on the thigh, the knee being raised slowly five or six centimeters. The toes were immovable. Pricking of the skin of the calf produced the same movement, as did also pinching of the tendo-Achillis and tapping of the crest of the tibia. This same response has been noted by Sherrington in monkeys with several cords, and is explained by him as a short-cut reflex when the long path is interrupted.

No disturbance of visceral sensibility could be detected. Though the skin of the scrotum was anesthetic, pressure on the testicles caused severe pain, referred to the corresponding side, not to the testicle, but rather to the skin of the lower abdomen. The ureter and bladder were normally sensitive to pain.

When paraplegia instantly follows injury, as in this case, the inference is that the cord has been injured or compressed by a hemorrhage. Yet this rule is not absolute, and Pitres recalls several cases where recovery followed when not expected. Indeed, shock alone may suffice instantly to bring about a paraplegia after an accident. Moreover, a partial lesion at the time of the accident may appear complete. Time is required to settle the diagnosis; and if the paraplegia is not only complete, but also permanent, the cord is doubtless severed.

As to the reflexes, the early statement of Bastian, that when the cord is cut, all the reflexes, instead of being exaggerated, are abolished permanently, though received at first with marked incredulity, is now almost universally accepted. The present case substantiates this affirmation. Never has there been an exception to this law.

The prognosis of such lesions is very bad on account of bed-sores and cystitis. Very few survive even four or five months. The treatment must needs be palliative, special attention being given bed-sores and bladder. The author's conclusions follow:

(1). Complete transverse section of the spinal cord in the middorsal region causes complete loss of general sensation in the legs and the skin of the trunk below the site of the injury; but visceral sensibility is not affected, especially the sensations of hunger and thirst, of fulness or emptiness of the stomach or bladder, deep epigastric sensibility to percussion, and sensitiveness of the testicles on pressure. (2) Such transverse section of the cord is accompanied by a total loss of muscular tonus and of all of the cutaneous and deep reflexes of the lower extremities. Occasionally slow movements of flexion of the leg on the thigh and raising of the knee above the level of the bed may be brought about by irritation of the calf; and occasionally spontaneous jerkings of the muscles take place involuntarily, without the patient's knowledge. These are doubtless due to direct irritation of the distal end of the severed cord. (3) A flaccid paraplegia immediately following trauma to the spinal column, with loss of cutaneous and tendon-reflexes, does not justify the diagnosis of severing of the cord. To make this diagnosis it must be known that the lesion is permanent, and time is required to make this discovery.

TUMORS OF THE SPINAL CORD.

Unusual Localization. Roque, Chalier, and Gignoux[1] report a case of a tumor originating in the meninges at the front end of the foramen magnum, and compressing the upper end of the cord and the lower end of the medulla. Such a location is very rare indeed, a case having been published by Chavigny and one by Raymond in his *Cliniques,* one by Dercum of chondro-sarcoma of the occipital bone, and finally one by Lannois and Perret. The case herewith reported resembled Raymond's case. The patient was a woman, 61 years of age, previously healthy, suddenly seized with persistent occipital headache. A month later pains in the lower extremities began abruptly, especially in the joints; but these were not swollen or red. Walking became painful, finally impossible; and because of the loss of strength in legs and arms, the patient was obliged to take to her bed. Every muscle in her body except those supplied by the cranial nerves was paralyzed, yet irritation of the soles of the feet or the toes led to a withdrawal of the legs. *Sensation was in every respect intact.* The reflexes were present, without diminution, in both the lower and upper extremities. The Babinski toe-sign was sometimes present, and sometimes absent. The pupils were equal, contracted, but reacted to light and accommodation. There was no disturbance of intelligence or of the emotions, of pulse or respiration. The condition lasted a few days, when the bladder began to overflow spontaneously. Fever then appeared. Lumbar puncture was made and 10 c.c. of absolutely clear fluid were withdrawn. Subsequently the temperature became higher, and death followed.

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(1) Lyon Méd., July 19, 1908.

Postmortem examination showed a tumor compressing the lower part of the closed medulla and the top of the spinal cord. The tumor straddled the anterior margin of the foramen magnum, and was approximately 1½ centimeters thick and 2½ broad. Above it reached the beginning of the basilar groove, and below, the anterior arch of the atlas and the odontoid process of the axis. It clearly sprang from the meninges, and did not adhere to the nerve tissue. It compressed the cord at the exact level of the foramen, softening it for a vertical distance of ¾ of a centimeter. The spinal and accessory portions of the spinal accessory and the hypoglossal nerves wholly escaped.

The main symptom in the case, an absolute quadriplegia, is explained by the involvement of the front of the cord and medulla. The absence of sensory disturbances is explained by the location of the tumor. Curiously enough there were no signs on the part of the pupils except possibly a negligible narrowing, no signs of respiratory or circulatory disturbances, such as bradycardia or the Stokes-Adams syndrome. Several cases of bradycardia have been reported in the literature and attributed to narrowing of the foramen magnum.

Surgical Treatment. H. Oppenheim and M. Borchardt[1] report another case of tumor of the cord successfully removed.[1] The patient was a young man, 23 years of age. The onset was with pains in the left buttock, soon becoming persistent and violent, followed by paresis of the left leg, numbness, and disturbance of gait. Later there ensued loss of control of the sphincter ani with preservation of bladder control. The physical findings are summarized in Oppenheim's report to Borchardt's surgical clinic: "In this patient I find the following: Spastic paresis of the left leg. In the right there are only spastic phenomena present in slight degree, but in the left leg the paresis is considerable. Moreover, in the left leg there is a special paresis of the quadriceps extensor of nuclear or radicular origin; that is, the muscle is atrophic with reduction of the electrical excitability, and the knee-jerk is weakened as compared with the right, and directly contrary to the spastic condition in which the left leg is. Besides, there are rather marked disturbances of sensation, such as slight tactile losses in the territory of the internal saphenous nerve, and very marked bilateral losses of the pain and temperature sense in the feet and calves. Lastly, there are mild disturbances of the bladder and rectum. There is nothing pathologic in the spinal column.

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[1] Berl. klin. Woch., Sept. 9, 1907.

"These symptoms indicate a process in the territory of the lumbar portion of the cord, at the point of origin of the III. and IV. lumbar roots, exerting its chief effect on the left half of the cord.

"The process can be one of the following: (1) Caries. (2) Extramedullary tumor. (3) Intramedullary tumor. The complete absence of spinal column symptoms speaks against caries. The question as to whether it is an intramedullary or extramedullary tumor cannot be answered with certainty. The bilateral distribution of the sensory disturbances with the essentially unilateral distribution of the motor disturbances argues in favor of an intramedullary tumor, while the pronounced development of the spasticity indicates rather an extramedullary growth. All things considered, it is best to perform an exploratory laminectomy later on, about the level of the XII. dorsal vertebra, but I would recommend it to be deferred until the patient has been under observation in your clinic for some time, and after repeated examination."

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(1) See Practical Medicine Series, Vol. X, 1906, p. 200.

The patient having grown worse, the question of operation was referred to him. He at once declared in its favor and Borchardt performed it. A tumor about an inch long and one-half to five-eighths of an inch wide was found on the anterior left side of the cord, compressing it. It was easily removed and the patient is slowly regaining his normal condition. This is Borchardt's fourth case of true spinal cord tumor upon which he has operated, and all four have been successfully removed.

Cyst in the Spinal Canal. W. C. Krauss, of Buffalo,[1] reports a case of cyst within the spinal canal, removed by Roswell Park, and resulting in recovery. A diagnosis of spinal cord tumor was definitely made and properly localized. At the operation a cyst was found, the exact nature of which was not disclosed, "but it seemed evident that there was a cavity with eroded walls connecting with the spinal canal, containing mostly fluid, with a little semisolid tissue material."

Krauss has gathered eleven cases of cyst of the spinal canal pressing upon the cord, and of these, seven terminated in recovery. All were extradural except two. The editor of *Brain* appends six other cases to the list given by Krauss, taken from English medical literature, all six resulting in recovery, ranging from slight to complete. There are thus only 4 fatal cases in 17 which have been operated upon.

A slightly similar case is reported by W. B. Warrington and K. W. Monsarrat.[2] The chief symptom was paraplegia due to an intramedullary lesion, and was treated with some success by the removal of a local accumulation of fluid. "The conditions causing a slowly increasing paralysis of the lower limbs are not numerous, and when the more common diseases such as disseminated sclerosis, amyotrophic lateral sclerosis, syringomyelia and the results of tubercle and syphilis are excluded, the diagnosis must be carefully considered, for the cause may be one of those conditions in which surgery affords the best or only chance of remedy. Tumors of the meninges are naturally the cases in which the most brilliant results have been ob-

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(1) Brain, Part IV, 1907.
(2) The Lancet, Jan. 11, 1908.

tained. The essential symptom is the combination of pain limited to definite regions of root distribution with paralysis indicating an advancing transverse lesion of the cord. Even should pain be absent, a progressive palsy indicating interference with conduction at some level of the cord should suggest the presence of a tumor-like growth.

"Lesions and growths within the medulla spinalis itself are considered inoperable. This case is recorded as an instance in which a focal intramedullary lesion was diagnosed and found and in which the removal of about 2 cubic centimetres of fluid was followed by great relative improvement in the patient's condition. The pathology of the case is not demonstrable, but the history and symptoms lead me to believe that the original lesion was a glioma of the cord and its etiology connected with an injury.

"The patient, a man aged 22 years, gave the following essential history: "Injury to the lower part of the spine followed by pain in the lower limbs and trunk for 2 years; a slow and slight weakness in the legs becoming worse after a period of rest in bed and, after an attack of pneumonia, a gradual recovery, then a sudden complete paralysis of the right leg and a gradual paralysis of the left, leading to complete paraplegia.

"Both lower limbs were absolutely powerless, with the exception of a very feeble flexion of the thighs. The limbs were flaccid; the feet and legs were blue and cold. On the inner side of each great toe there was a small sore. The muscles were flabby and the right limb, in comparison with its fellow, was wasted. Electrical irritability was normal both to faradic and galvanic excitation, though somewhat diminished in the right side. Both knee-jerks were extremely feeble, so that it was doubtful at times if the left could be obtained. The ankle-jerks were absent; the right plantar response was of a feeble extensor type, the left was not obtained. Occasionally involuntary spasmodic flexion movements of the legs and thighs occurred. Abdominal, epigastric, and cremasteric reflexes were not obtained. The diaphragm and intercostal muscles acted well. The cranial nerves and the upper limbs were normal. The spine was carefully examined without detecting any deformity, but pressure over the ninth to twelfth dorsal spines caused pain round the trunk at the level of the last rib on both sides. Flexion of the spine also caused some pain about the mid and lower dorsal region. Retention of urine was present on 2 occasions, once persisting for 86 hours.

"With regard to sensation, a fairly horizontal line starting posteriorly from the ninth dorsal spine or passing anteriorly through the lowest part of the ensiform cartilage and thus corresponding to the seventh dorsal spinal segment, formed the upper limit of a fairly well-defined loss of sensibility. Below this, pain from a pin prick and the recognition of heat or cold were absent, but on the right side over the leg and thigh a touch by cotton wool was felt, as well as the pressure produced by the head of a pin. On the left side both these forms of sensation also were absent. The patient therefore presented the signs of a transverse lesion of the cord at about the mid-dorsal region, the absolute loss of power, great loss of sensation, and the feebleness of the reflexes showed that conduction was greatly interfered with, and if the disease progressed a complete severance of the brain from the lower part of the cord was to be expected.

"In *diagnosis,* system disease, disseminated sclerosis, and the results of acute myelitis or myelomalacia could be excluded. There remained to be considered some form of pressure paraplegia, syringomyelia, and late effects of trauma. It was felt that a study of the history could alone give any indication as to the nature of the lesion and it will be remembered that sudden paralysis of one limb (the right) was a striking feature. Though a sudden paraplegia not infrequently occurs in the course of tuberculosis of the spine its cause is either an increase in the pressure or a softening of the cord itself; it appeared unlikely that either of these conditions would account for a sudden hemiparaplegia. On this account, together with the absence of local signs in the bones or of constitutional symptoms, caries was excluded. New growths and other forms of pressure had therefore to be considered. The absence of pain was against the diagnosis of extramedullary lesion, though, as already mentioned, not conclusively so. The sudden right hemiparaplegia indicated a vascular origin as its cause, and together with the subsequent slow paralysis of the left limb suggested an intramedullary growth, and this was thought to be a glioma. The hypothesis of hemorrhage into a gliomatous tumor or into a cavity in the cord seemed the best explanation of the facts of the case, and the findings at the operation are consistent with this view."

Operation was performed by Dr. Monsarrat, the 4 dorsal spines from the sixth to the ninth vertebra being removed. The dura mater was abnormally vascular, and was intimately adherent to the cord, especially at the level of the seventh dorsal vertebra. There was no escape of cerebrospinal fluid. The cord bulged through the opened dura and showed the posterior columns to be transformed into a grayish-black jelly-like substance. At the lower end of this area a fine knife was inserted and about 2 cubic centimeters of a clear yellow fluid escaped, and the protrusion collapsed.

The patient improved continually after the operation, regaining the ability of walking 40 yards with crutches without a rest. The right leg is stronger than the left. He is able to rise from the sitting to the erect position. The nutrition of the muscles is good, but the feet are still blue and cold. There is ankle-clonus on the right side, and plantar extensor response (Babinski toe-sign) on both sides. The abdominal, epigastric, and cremasteric responses are absent. There is no bladder trouble. Sensation is much improved, but "spot naming" is inaccurate. "On the left side these forms of sensibility are very deficient, but there is still some power of appreciating pressure by the head of a pin from that of the point, but no pain arises. The upper limit of loss of sensation is at the original level and there is now a zone on the trunk and outer sides of right thigh corresponding to representation of the seventh dorsal to second lumbar segment, on which light touch is not recognized nor a prick appreciated as such. Thus sensation has greatly improved in the right leg and thigh, but has diminished in degree over an area which corresponds to the extent of the surgical procedure."

SYRINGOMYELIA.

Unusual Cases. (a) J. Mollard and J. Chattot[1] report a case of syringomyelia associated with symmetrical lipomatosis. The syringomyelia developed first, the scoliosis having existed 2 years before the onset of the tumors. The authors are inclined to believe that syringomyelia counts among its trophic disturbances the development of lipomatous masses more or less symmetrical, though such an observation has never before been made. A case of symmetrical lipomatosis and intermittent claudication was reported by Cade, in 1907, however, and he inclines toward the view that lipomatosis has a trophoneurotic origin. Certainly the old idea that such tumors have a lymphatic origin is untenable, as careful histological examination shows no lymphatic tissue within them.

(b) R. Milchner[2] reports very carefully a case of syringomyelia which he regards as noteworthy on account of the great extent of the process and its practical limitation to one side of the body. The process, beginning in the right facial muscles, in time involved the right leg, and after several years of quiescence, the right shoulder-girdle. Apparently the onset was in very early youth, for the facial paresis began during his school days. After a pause of many years, possibly because of over-exertion during a week of marching, the process began again in the right calf. After another long pause, the right shoulder has become involved. There must be, then, 3 distinct foci in the cord.

MULTIPLE SCLEROSIS.

Symptomatology. R. T. Williamson[3] reports 4 cases of multiple sclerosis beginning with failure of vision. "E. Müller states that in a young person who has never suffered from syphilis, lead poisoning, or alcoholism, if amblyopia should suddenly develop in one eye and then partially subside, and if some time later a similar affection should develop in the other eye, the diagnosis of disseminated sclerosis is very probable. In such a case confirma-

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(1) Lyon Méd., May 10, 1908.
(2) Berl. klin. Woch., April 6, 1908.
(3) The Lancet, May 2, 1908.

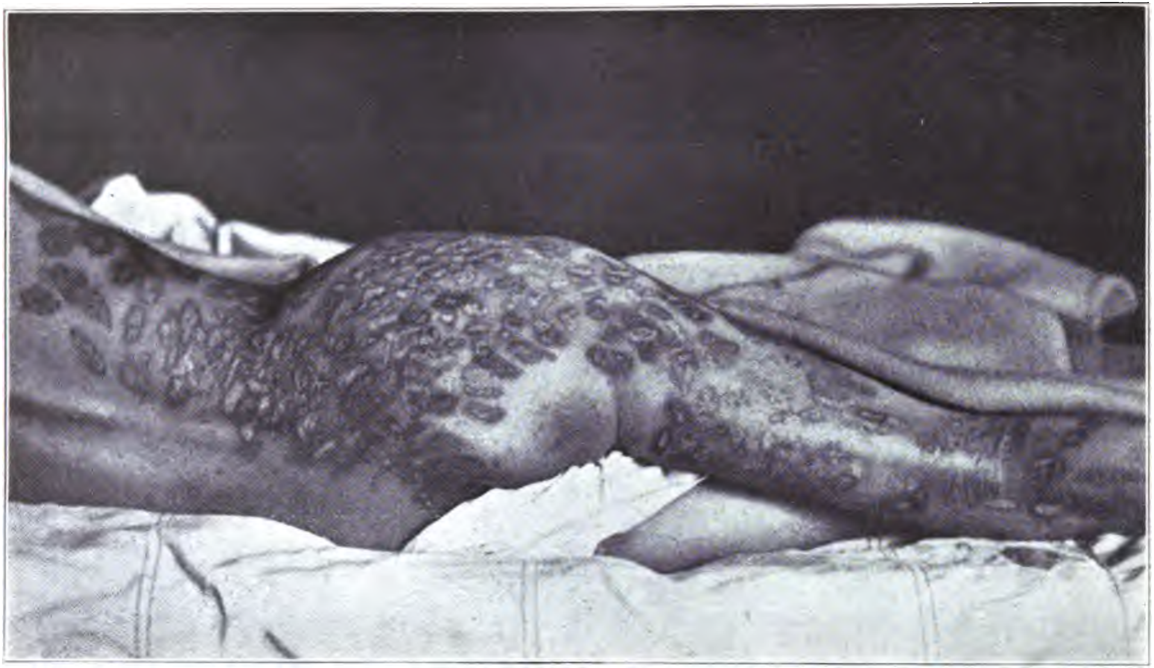

PLATE V.

Rash in multiple sclerosis.—J. A. Ormerod (page 153).

tory signs would be—pallor of the temporal half of the optic disk, with the characteristic relations of the field and defect of vision, failure of the abdominal reflexes, the Babinski extensor type of plantar reflex, and slight shaking of the hands on fine movements. The age of the patient (from 20 to 40 years), the absence of any recognizable cause, the absence of pain (in most cases), and the satisfactory general condition would be points in favor of the diagnosis of disseminated sclerosis."

With these statements Williamson agrees, and his 4 cases, together with 2 quoted from memory, substantiate Müller's claim.

"In the form of disseminated sclerosis beginning with visual failure, pain and anesthesia are very rare, as in other forms of this disease. The signs of chief diagnostic value at the early stage are: (1) unilateral or bilateral visual failure, with central scotoma in some cases and often with pallor of the optic disk, especially of the temporal half; (2) the Babinski type of plantar reflex on one or both sides; (3) the irregular and shaky character of the handwriting, even when the tremor is so slight that it can hardly be detected (a most valuable sign); and (4) the age of the patient, under 40 years. The absence of any cause for the affection and of any history of syphilis, the absence of pain and anesthesia, and the presence of the knee-jerks, tendo-Achillis reflexes, and pupillary reflexes are points of diagnostic value in favor of disseminated sclerosis. It is interesting to note that in many cases after the visual defect has become marked there is a decided improvement, or almost complete recovery of vision. In other cases the visual defect remains more or less stationary, and when it does progress it very rarely advances to complete blindness, in this respect differing from the optic atrophy of tabes dorsalis."

Diagnosis. J. A. Ormerod[1] presents 2 cases of multiple sclerosis with autopsy. In the first case the disease was not diagnosed during life, nor even suspected. Indeed, there were no facts to suggest it. The leading symptom on admission to the Queen Square Hospital was an intense flexor spasm of the legs. This symptom is of course seen in long-standing cases of multiple sclerosis, but the patient had been ill only 10 months. Summarized, the case is as follows: "Onset sudden, 10 months before admission. -Extensive spasms of the legs, with diarrhea, repeated on 4 successive days. Further cramps in the legs 5 weeks later, with pain, finally assuming the form of flexor spasms, lasting for some months. Gradual development of flexor contraction of the lower limbs. Painful tinglings in the legs 6 weeks after the onset; incontinence of urine, with anesthesia of the bladder. Rigidity of abdominal muscles 2 months before admission. Pain in back and lower limbs.

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(1) Brain, Part III, 1907.

"On admission, the lower limbs were firmly flexed at hip and knee; there was much tenderness to touch or manipulation; there was incontinence of urine. The legs straightened under chloroform 2½ months after admission. The patient died of exhaustion some 9 days later. Postmortem, the viscera were found normal. Plaques of disseminated sclerosis were found in the spinal cord, principally in the cervical region, pons, medulla, right optic thalamus, and right optic tract."

The following is the summary of the second case: "Woman of middle age, who had had many illnesses. Lymphangitis with abscesses of right upper limb; splint applied; stiffness of elbow and fingers followed, and paresthesia in fingers, spreading to right side of trunk. Afterwards abscess right thigh, weakness right lower limb; peculiar rash right thigh and right side of body. On admission to St. Bartholomew's (January 13, 1905), 3 or 4 years after onset, she was completely bedridden. Right upper limb was rigid, and particularly the fingers, where there was great pain on passive movement; right hip rigid, but less so than upper limb; wasting of right limbs, especially the upper limb; right hemianesthesia, and a rash of peculiar appearance and distribution. Plantar reflexes extensor; optic atrophy (observed later). During next few weeks much improvement in motor condition of right limbs; slight spread of the eruption.

"Transferred to Queen Square Hospital 4½ months later (May 29). Still extreme rigidity of right fingers; anesthesia has spread upwards to right clavicle; rash now stationary. No further change for the next 3 or 4 months, then (September-October, 1905) spread of symptoms to left side, viz., anesthesia, left as well as right, from neck downwards, weakness of left limbs, jumping movements, and disagreeable sensations in left limbs; further slight spread of rash; general debility and depression. She left Queen Square at Christmas, 1905, and died of abdominal cancer in November, 1906, some 5 or 6 years after the onset. Examination of the nervous system showed the presence of disseminated sclerosis." Plate V. shows the remarkable rash which was present.

Kuckro[1] reports an interesting case under the title: *Multiple Sclerosis or Cerebrospinal Syphilis.* [See Spiller's case, Practical Medicine Series, Vol X, 1907, p. 159.] The patient, a young man, had been infected with syphilis 8 years before, and had been carefully treated. There now appears an acute paretic and ataxic disturbance of both legs, with weakness of the bladder, these symptoms gradually deepening. At present there are to be found spastic-ataxic paresis of both legs, increase of the reflexes with the Babinski toe-sign, absence of the abdominal reflex, normal pupillary reflexes, right-sided hemihypesthesia with disturbance of coördination of the muscles of the right hand, absence of taste, smell, and hearing on the right side, pallor of the temporal halves of both papillæ, on the right a central color scotoma and on the left a sector-shaped scotoma. Treatment with mercury and potassic iodid, with rest, in a few days' time restored his gait almost to the normal, except that when he was tired the spastic-ataxia paresis reappeared. The cerebrospinal fluid showed no increase of albumin, no lymphocytosis, and no cells at all. There were no psychic or hysteric symptoms.

The sudden onset is suggestive of cerebrospinal syphilis, though multiple sclerosis may begin abruptly. As to the above symptoms, practically all occur in multiple sclerosis, and in cerebrospinal syphilis. The central color-scotoma is not uncommon in multiple sclerosis, and the absent abdominal reflex speaks in favor of that disease. More important than these differential factors, however, is the absence of lymphocytosis of the cerebrospinal fluid. Moreover multiple sclerosis best accounts for the hemiplegic distribution of the sensory losses. The case at least shows,

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(1) Muench. med. Woch., Nov. 5, 1907.

no matter whether it be multiple sclerosis as the author calls it, or cerebrospinal syphilis, that the differential diagnosis between these two diseases is sometimes very difficult.

Locomotor Ataxia or Tabes Dorsalis.

Pathogenesis. The pathogenesis of tabes dorsalis is ably discussed by T. A. Williams,[1] who supports Nageotte's theory. At the very beginning of this paper he hastens to state that he rejects the usual explanation that tabes is a primary dystrophy of the posterior columns or of the sensory protoneurones, as a whole or an atrophy thereof, directly caused by alcohol, prolonged strain, syphilis or sexual excesses. The author first calls attention to the well-known physiological fact that the posterior ganglion cell is capable of exercising a regenerating function over its axones, only when the fiber concerned possesses a neurilemma, while within the spinal cord the axone has no sheath, being provided with only the white substance of Schwann. Hence if the function of this neurone is impaired the peripheral sensory nerve may regenerate, but the fiber in the cord will be destroyed permanently.

Williams next recalls that between the lower spinal ganglia and the segments of the spinal cord corresponding with each, there intervene several inches, which the radicular nerve traverses in contact with the leptomeninges, as

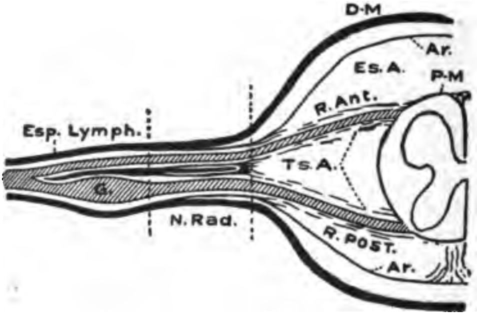

Fig. 1.—Diagram showing the relations of the meninges to the radicular nerve. *N. Rad.*, radicular nerve ; *PM*, pia mater ; *Ar.*, arachnoid ; *DM*, dura mater ; *R. Ant.*, anterior roots ; *R. Post.*, posterior root ; *G*, ganglion ; *E. L.*, facultative lymphatic space. (From Nageotte.)

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(1) Am. Jour. Med. Sciences, August, 1908.

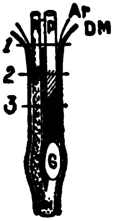

Fig. 2.—*AP,* anterior and posterior roots; *Ar.,* arachnoid; *DM,* dura mater; *G,* intervertebral ganglion. (From Nageotte.)

is shown in Fig. 1. This shows clearly the canal formed by the pachymeningeal reflection as the nerve pierces the sheath to become peripheral. In this canal it is probable that communication takes place between the cerebrospinal fluid filling it and the lymph in the sheath of the peripheral nerve. Figs. 2, 3, 4, and 5 show the lesions described by Nageotte in this locality in cases of tabes and tabo-paralysis.

Now, even though the infection is very chronic, and the reaction of the granulomatous process is comparatively indolent, nevertheless in this situation they are sufficiently serious to affect the nerve fibers with which they are in contact; for in the circumscribed canal of unyielding dura mater, the pressure of such inflammatory tissue and exudate, either destroys the nerve fibers, or at least sufficiently injures them to prevent the passage of impulses from the ganglion-cell. And since this ganglion-cell exerts a trophic influence over its fiber, its fiber degenerates. Having once degenerated, and possessing no neurilemma, it can never regenerate, even after the granuloma has undergone absorption; and the postmortem appearance of tabes is thus constituted. Though Nageotte was of the opinion that a toxic influence is exerted upon the nerve-fibers by the process, the mechanical factor alone is sufficient to account for the pathologic findings of tabes.

"Figs. 3 and 4 show how disparate is the involvement of the radicular nerve. Whether this is due to an elective affinity of a toxin for a particular sensory system, or whether it is due to an accidental incidence depending on the local differences of arrangement, cannot be affirmed with certainty." Clinical evidence seems to show that the

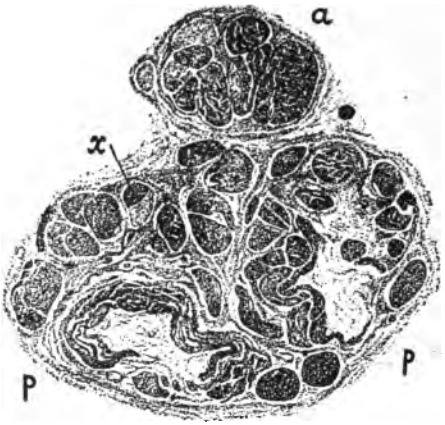

Fig. 3.—Cross-section of the radicular nerve in a general paralytic in which the lesions of the anterior root (*a*) were more prominent than those of the posterior root (*p*). The section is at line 3 of Fig. 2 in which the posterior root is involved also. Note the perineuritis with its cavity formation, and the paler bundles of degenerated fibres even where there is no perineuritis. The bundle *x* is half intact. (From Nageotte.)

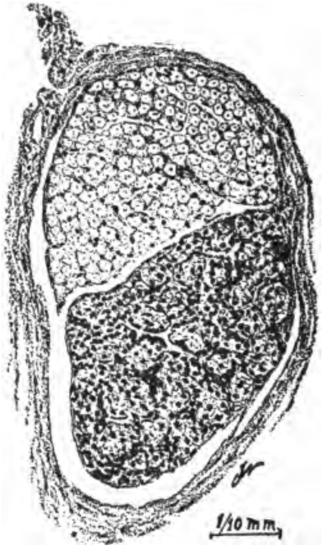

Fig. 4.—The region *x* in Fig. 3 magnified 90 diameters to show the healthy fibers and those from which the myelin has disappeared. (From Nageotte.)

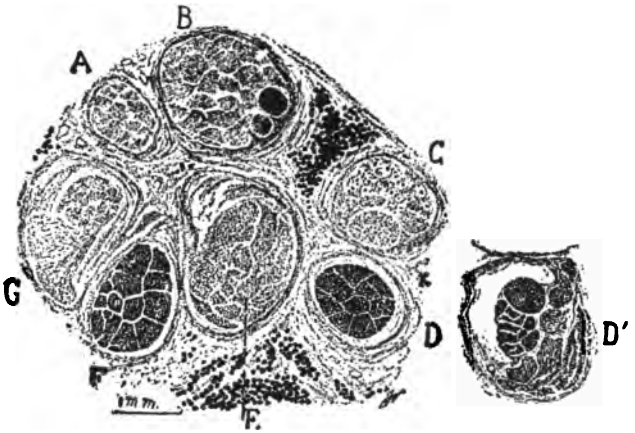

Fig. 5.—Cross-section of the radicular nerve in a tabetic, showing the round-cell infiltration. The bundles *F* and *D* are spared, while all the rest are degenerated. *D'* is a cross-section of *D* lower down. Notice the area of lacunar perineuritis which displaces some fasciculi. Lower still this necrosis was found to occupy the center of the bundle. (From Nageotte.)

fibers carrying the so-called muscle and joint-sense impulses are early involved, while those subserving the sensation of cold are spared relatively long.

If Nageotte's view is correct, both motor and sensory fibers should be involved in tabes, and both are involved. Nageotte's sections show that the anterior roots often degenerate, and indeed muscular atrophy is much more common in tabes than earlier writers allowed. But the anterior roots regenerate effectively and early because, possessing a neurilemma, such regeneration is the physiologic law. On the other hand, the sensory root fibers entering the cord, having no neurilemma in the cord, will not regenerate; hence tabes is apparently a sensory disease.

The author's conclusions follow: "1. Tabes dorsalis is a secondary degeneration in the posterior columns, due to a chronic meningitis, very probably of syphilitic nature. 2. The arrangement of the meninges surrounding the radicular nerve renders it peculiarly susceptible at that spot to mechanical or toxic injury. 3. The unequal incidence of the affection upon different fibers of the posterior root is probably due to unascertained peculiarity of structure or arrangement of fasciculi, rather than to any selective toxic influence. 4. The lesions tend toward resolution and arrest, even though the process may continue during the life of the individual. 5. With this arrest, regeneration tends to occur in the radicular nerve, the amount in the anterior root being relatively considerable, while that in the posterior root is less in amount and functionally insignificant, as a rule. 6. The otherwise inexplicable vasomotor and cranial nerve symptoms and postmortem findings in this disease are shown thus to be necessary concomitants of the tabetic process. 7. The question of the pathogenesis of the polyneuritic manifestations found in tabetics is not yet answered."

Attention is called by C. D. Camp[1] to *motor paralysis as an early sign of tabes dorsalis,* and reports 2 cases from the service of W. G. Spiller at the Polyclinic Hospital in Philadelphia. In the first patient there was a decided toe-drop on the right side, and in the second loss of power in the dorso-flexors of the left foot. In each of these cases multiple neuritis was excluded, the entire symptomatology in each case being due to tabes alone. [The above cases have a bearing on Nageotte's theory of the pathogenesis of tabes.—Ed.]

"The question of the location of the lesion in these cases of early paralysis in tabes dorsalis cannot be regarded as definitely settled. By some it will be held that the lesion is a motor neuritis due to the action of the mysterious toxin that causes tabes, but acting more extensively, and on the motor as well as on the sensory neurone. A second view is that the paralysis is due to an affection of the motor cells of the anterior horns of the spinal cord, not perhaps as severe as those causing the amyotrophies, and due to a propagation of the lesion across the posterior cornua to the motor cells. Whether the lesion were in the motor nerves or in the motor cells, if it were severe the paralysis would be followed by atrophy and changes in the electrical reactions such as occurred in the second case."

Etiology. (a) *Syphilis.* Adolf Strümpell[2] calls attention to the relationship between tabes dorsalis and dis-

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(1) Med. Record, Nov. 23, 1907.
(2) Deut. med. Woch., Nov. 21, 1907.

eases of the heart and great vessels. In patients with aortic insufficiency, sclerosis of the aorta and aortic aneurysm not infrequently signs of a concomitant tabes may be found if specially looked for, such as absence of the pupillary light-reflex, absent tendon-reflexes, or lancinating pains, etc. And very often well marked cases of tabes will show, when carefully examined, signs of aortic insufficiency and aortic sclerosis. These combinations of apparently two different sorts of pathologic processes depend upon the fact that they are due to the same cause, for they are both, according to Strümpell, due to syphilitic infection. The establishment of a single tabetic symptom, especiallly of the absence of the pupillary light-reflex, may be looked upon as a stigma of syphilis, and is therefore of special diagnostic value.

In a paper on the relation of locomotor ataxia and paresis, J. Ramsay Hunt[1] says: "In conclusion, it may be said that tabes dorsalis, dementia paralytica, and the combined form of the two affections, all have the common ætiological factor of an antecedent syphilis. The pathological alterations in the cerebral cortex and the spinal cord are essentially the same in both the isolated and the combined forms. The clinical combinations of tabes and paresis are so varied and so numerous that a gradual transition may be traced from locomotor ataxia, on the one hand, to general paralysis of the insane on the other. In fact, it may be said that the more our knowledge of these parasyphilitic affections of the brain and spinal cord increases the more significant appears their combination, and the stronger becomes the tendency to regard them all as essentially of the same nature and origin."

(b) *Trauma.* According to L. Feilchenfeld,[2] tabes and progressive paralysis also may stand in a fairly close relation with trauma. The latter may (1) cause an existing disease to enter upon a more serious phase; (2) or it may lead to an unusual complication; or (3) it may result fatally much sooner than it otherwise might. Thus after an accident, a tabes which had previously been latent, may become abruptly evident, or a complication such as arthropathy, or cystitis, or optic atrophy, may set in; or there may be a sudden exacerbation of the case with a comparatively rapid course and early death.

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(1) New York Med. Jour., July 4, 1908.
(2) Berl. klin. Woch., Feb. 3, 1908.

In order that a trauma may stand in any causal relation with tabes, it must be (1) a fairly pronounced injury; (2) the site of the trauma must be such as will affect brain or cord; (3) there must be a direct sequence in point of time between trauma and symptoms. To illustrate these points Feilchenfeld instances 17 cases of tabes, very briefly recounting injury and effect.

(c) *General Remarks.* Byrom Bramwell presents an analysis of 263 cases of tabes dorsalis,[1] which he has met with in hospital and private practice. As far as possible he has followed out all of these cases, and has thus been able to trace the course of the disease during a period of several years in a considerable proportion of the 263 cases; and in most fatal cases he has been able to give the total duration and the immediate cause of death.

As to type of the disease, 192, or 73 per cent., were in the ataxic, and 71, or 22.9 per cent. in the preataxic stage; 239, or 90.8 per cent., were males, and 24, or 9.1 per cent., were females. The age at onset in 258 of the cases is shown in Table 1.

TABLE I.—*Age at Onset in 258 Cases of Tabes.*

Age.	No. of Cases.	Per Cent.
From 20 to 30 years	37	14.3
From 31 to 40 years	121	46.8
From 41 to 50 years	70	27.0
From 51 to 60 years	25	9.6
Above 60 years	5	1.9
Total	258	99.6

As to etiology, 155, or 61.2 per cent. of the 253 cases, admitted syphilis; in 12 cases, or 4.7 per cent., it was practically certain—from the family history of repeated miscarriages, stillborn children, husband syphilitic, etc.—that the patient had had syphilis; in 16 cases, or 6.3 per cent., the history as regards syphilis was doubtful; and in 70 cases, or 27.6 per cent., the patients denied syphilis. In 42 cases in which patients denied syphilis, or in which syphilis was doubtful, gonorrhea was admitted. "Consequently, in 197 of the 253 cases there was a definite history of some form of venereal disease, and in 12 other cases it is practically certain that the patients had had syphilis. It is, therefore, practically certain that in 209, or 82.6 per cent., of the 253 cases the patients had had some form of venereal disease. In 1 case in which syphilis was denied the condition of the teeth suggested congenital syphilis.

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(1) Brit. Med. Jour., March 21, 1908.

"Further, it is, of course, certain that in some (perhaps a considerable proportion) of the 70 cases in which the patients denied syphilis, they had actually suffered from that disease. In several of the cases in which syphilis was ultimately admitted the patients had, at first, emphatically denied that they had suffered from the disease. In several cases the syphilis had been treated very thoroughly for long periods of time with mercury. These cases show—and this is an important practical point from an insurance point of view—that prolonged and thorough mercurial treatment does not, in all cases at all events, prevent the subsequent development of tabes."

The length of time between the occurrence of the venereal sore and the first symptoms of tabes was determined in 141 cases.

TABLE II.—*Time between the Chancre and the First Symptoms in 141 Cases of Tabes.*

Age.	No. of Cases.	Per Cent.
Under 1 year	1	0.7
1 to 5 years (inclusive)	26	18.4
6 to 10 years (inclusive)	36	25.5
11 to 15 years (inclusive)	30	21.2
16 to 20 years (inclusive)	33	23.4
21 to 25 years (inclusive)	11	7.8
26 to 30 years (inclusive)	2	1.4
36 to 40 years (inclusive)	1	0.7
41 to 45 years (inclusive)	1	0.7
Total	141	99.8

Sexual excess may be a contributing factor in the etiology, but was not inquired into sufficiently to make any conclusion of value. In 7 of the 263 cases, marriage seemed to have a decided influence either in producing or aggravating the disease. Alcoholic excess was admitted in only 26 cases; the actual number was probably much larger. In 7 cases an injury or operation was regarded by the patient as the cause of the disease, but it is very doubtful if in any of these cases the injury was the real cause. Exposure to cold and wet, lying out at night, mental worry, infectious diseases, and lead poisoning were all given by patients as the cause of their trouble.

Symptomatology. The nature of the first symptoms was determined in 258 of the 263 cases.

TABLE III.—*Nature of the First Symptoms, as stated by the Patient, in 258 Cases of Tabes.*

Nature of Symptom.	Cases.	Per Cent. of 258 Cases.
Lightning pains (alone)	133	51.5
Diplopia	20	7.7
Dimness of vision	20	7.7
Ataxia	17	6.5
Lightning pains and ataxia	14	5.4
Derangement of urination	10	3.8
Gastric crises	6	2.3
Numbness (in feet, etc.)	5	1.9
Pains and loss of vision	3	1.1
Ataxia, pain in back, and numbness in feet	2	0.7
Vomiting (not due to gastric crisis)	2	0.7
Pains and giddiness; diplopia and giddiness; derangement of urination and debility; pain in the back; pain in the back and ataxia; coldness of feet; weakness; ataxia and loss of sexual desire and power; ataxia and bladder trouble; lightning pains and bladder trouble; joint affection; numbness in the feet and catching of the toes; pain in the back and bladder trouble; ataxia and cramps in the hands; ataxia and girdle sensation; noise in the ear (like a singing bird); loss of sexual power and desire; numbness and cough; anesthesia of thorax; tired feeling in legs; pains, bladder trouble, and weakness in legs; inordinate sexual desire; pains, ataxia, and bladder trouble; pains and numbness; pains and girdle sensation; loss of vision and giving way of legs; 1 case each	26	10.0
	258	99.3

An excellent summary of the symptomatology as to percentage is given in the following exhaustive table:

TABLE IV.—*Frequency and Percentage of Chief Symptoms in 263 Cases of Tabes.*

Nature of Symptom.	Frequency and Percentage of Different Symptoms.				
	In total 263 Cases.		In those Cases in which the Symptom was specifically Noted.		
	No. of Cases in which present.	Per Cent.	No. of Cases in which Symptom was Specifically Noted.	No. of such Cases in which Symptom was present.	Per Cent.
Lightning pains	241	92.2	263	244	92.2
Pain in the back	86	32.7	180	86	47.7
Girdle sensation	107	40.6	183	107	58.4
Paresthesiæ	178	67.6	214	178	83.1
Numbness	148	56.2	214	148	69.1
Coldness of feet	85	32.3	214	85	39.7
Pins and needles	49	18.6	214	49	22.9
Ground felt soft	53	20.1	214	53	24.7
Anesthesia in the legs	100	38.0	214	100	46.7
Anesthesia in the arms	51	19.3	174	51	29.3
Anesthesia in the trunk	77	29.2	175	77	44.0
Anesthesia in the face	8	3.0	159	8	5.0
Analgesia in the legs	107	40.6	196	107	54.5
Analgesia in the arms	96	36.5	176	96	54.5
Analgesia (ulnar)	54	20.5	103	54	52.4
Analgesia in the trunk	123	46.7	183	123	67.2
Analgesia (thoracic)	62	23.5	104	62	59.6
Analgesia in the head	4	1.5	157	4	2.5
Ataxic gait	192	73.0	263	192	73.0
Rombergism	190	72.2	262	190	72.5
Incoordination in the legs	184	69.9	259	184	71.0
Incoordination in the arms	58	22.0	214	58	27.1
Impairment of muscular sense in the legs	94	35.7	175	94	53.7
Impairment of muscular sense in the arms	26	9.8	153	26	16.9
Muscular analgesia in legs	81	30.7	132	81	61.3
Hypotonia in the legs	70	26.6	118	70	59.3
Paralysis, other than ocular	19	7.2	218	19	8.7
Loss of both knee-jerks	212	80.6	255	212	83.1
Loss of one knee-jerk	12	4.5	255	12	4.7
Loss of both Achilles-jerks	132	50.1	159	132	83.0
Loss of one Achilles-jerk	6	2.2	159	6	3.7
Loss of deep reflexes, upper extremities	62	23.5	131	62	46.9
Loss of jaw-jerk	30	11.4	116	30	25.8
Loss of plantar reflex	26	9.8	213	26	12.2
Babinski's sign present	7	2.6	135	7	5.2
No toe movement	17	6.4	135	17	12.5
Loss of cremasteric reflex	66	25.0	161	66	40.9
Loss of abdominal reflex	32	12.1	150	32	21.3
Derangement of bladder reflex	186	70.7	251	186	70.1
Derangement of rectal reflex	139	52.8	248	139	56.0
Derangement of sexual reflex	115	43.7	178	115	64.6
Pupils:					
Inequality in size	99	37.6	159	99	62.2
Contracted	117	44.4	208	117	56.2
Medium	69	26.2	208	69	33.1

Nature of Symptom.	Frequency and Percentage of Different Symptoms.				
	In total 263 Cases.		In those Cases in which the Symptom was specifically Noted.		
	No. of Cases in which present.	Per Cent.	No. of Cases in which Symptom was Specifically Noted.	No. of such Cases in which Symptom was present.	Per Cent.
Dilated	14	5.3	208	14	6.5
Irregularity of margins	32	12.1	68	32	47.0
Loss of light reflex	222	84.4	252	222	88.0
Loss of contraction on accommodation	37	14.0	252	37	14.6
Argyll-Robertson pupil	186	70.7	252	186	73.2
Optic atrophy	55	20.9	263	55	20.9
Ocular paralysis	38	14.4	263	38	14.4
Crises	35	13.3	222	35	15.7
Gastric	25	9.5	222	25	11.3
Rectal	6	2.2	222	6	2.7
Laryngeal	4	1.5	222	4	1.8
Vesical	2	0.7	222	2	0.9
Intestinal	2	0.7	222	2	0.9
Sexual	1	0.3	222	1	0.4
Nasal	1	0.3	222	1	0.4
Trophic lesions in bones and joints	17	6.4	240	17	6.8
Trophic lesions in skin and its appendages	19	7.2	240	19	7.9
Mental symptoms	27	10.2	263	27	10.2
General paralysis of the insane	30	11.4	263	70	11.4
Headache	31	11.7	188	31	16.4
Giddiness	33	12.5	186	33	17.7
Derangement of sleep	50	18.9	200	50	25.0
Impairment of hearing	25	9.5	187	25	13.4
Impairment of taste	2	0.7	179	2	1.1
Impairment of smell	2	0.7	178	2	1.1
Considerable loss of weight	78	29.6	224	78	34.8
Cardiac disease	14	5.3	234	14	5.9
Albuminuria	13	4.9	234	13	5.5

The immediate cause of death in 70 fatal cases is shown in Table V.—

TABLE V.—*Causes of Death in Seventy Fatal Cases of Tabes.*

General paralysis of the insane	15
General paralysis of the insane and albuminuria	1
General paralysis of the insane and pyelonephritis	1
Cerebral symptoms resembling acute general paralysis of the insane	1
Apoplexy	3
Exhaustion tabes	7
Exhaustion and bedsores	2
Exhaustion and gastric crises	1
Exhaustion, prolonged vomiting	1
Cystitis	2
Kidney disease, pyelitis	1
Diarrhea	1
Diabetes mellitus	1

Asthma	1
Phthisis	1
Phthisis and Pott's disease	1
Phthisis and morphin	1
Phthisis, ascites, etc.	1
Influenza and bronchitis	1
Pneumonia	2
Heart failure	1
Aneurysm, thoracic (one ruptured)	3
Aneurysm (abdominal)	1
Aortic regurgitation	1
Hemorrhage (? gastric)	1
Hemorrhage from bowel	1
Cut throat	1
Not known	16
	70

The total duration of the disease in these 70 cases is shown in Table VI:

TABLE VI.—*The Total Duration of the Disease in Seventy Fatal Cases of Tabes.*

Below 1 year	1 case
1 to 2 years	2 cases
2 to 3 years	2 cases
3 to 4 years	5 cases
4 to 5 years	9 cases
5 to 6 years	8 cases
6 to 7 years	3 cases
7 to 8 years	4 cases
8 to 9 years	3 cases
9 to 10 years	2 cases
10 to 11 years	8 cases
11 to 12 years	1 case
12 to 13 years	5 cases
13 to 14 years	1 case
14 to 15 years	1 case
15 to 16 years	3 cases
16 to 17 years	1 case
17 to 18 years	3 cases
20 to 21 years	1 case
21 to 22 years	1 case
22 to 23 years	1 case
Not stated	4 cases

Ocular Symptoms. The eye-symptoms of tabes dorsalis are elaborately considered by Massia and Delachanal.[1] After a brief historical review, they classify these symptoms as follows:

- A. Motor disturbances.
  - a. Extrinsic muscles.
  - b. Intrinsic muscles.
    - Iris.
      - Myosis or mydriasis.
      - Anisocoria (pupillary inequality).
      - Pupillary irregularity.
      - Atrophy of the iris.
    - Accommodation.
      - Argyll-Robertson sign.
      - Erb sign.
      - Pilcz sign.
      - Gowers sign.

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(1) Gaz. des Hop., Jan. 4 and 11, 1908.

B. Sensory disturbances.
  a. General sensibility.
    Superficial.
    Deep, or sensitiveness to pressure.
  b. Special sensibility or the visual sense.

C. Trophic, Vasomotor, and Secretory disturbances.
  Ocular hypotonia.
  Epiphora.
  Corneal herpes.
  Neuro-paralytic keratitis.
  Atrophy of the papilla.

Motor disturbances of the extrinsic muscles occur in nearly one-half of the cases, and as a rule come early in the course of the malady, in the preataxic stage. The muscles most commonly involved are the levator palpebræ superioris and the rectus externus. Very characteristic of these paralyses is their very abrupt onset and their short duration, for they usually last only a few weeks or even days. They are usually unilateral.

During the period of confirmed disease paralyses of these muscles also occur, but they are less frequent, of more gradual onset, and more persistent. There is ptosis and strabismus and the complaint of diplopia. This is homonymous when the false image is on the side of the paralyzed eye, and heteronymous when it is on the opposite side. The test is simple: place a lighted candle before the patient and a colored glass in front of one eye. The patient is then asked on which side of the uncolored image the colored one lies. If the images are crossed the diplopia is heteronymous. The rule is that homonymous diplopia results from paralysis of the external, and heteronymous from paralysis of the internal rectus muscles respectively.

Facial paralysis and paralysis of the sympathetic also occur, the former involving chiefly the orbicularis, and making it impossible for the lids to be perfectly closed, and permitting the lower lid to fall away from the eyeball, so that there is ectropion and epiphora. Consequent upon this motor defect is conjunctivitis, corneal ulcerations, and keratitis. Paralysis of the sympathetic leads to narrowing of the palpebral opening, and exophthalmia. The ocular hypotonia is doubtless due to paralysis of the sympathetic.

Of the intrinsic muscles, the iris alone bears the brunt of the tabetic attack. Myosis and mydriasis are both common, myosis being the rule and mydriasis the exception, myosis occurring in at least 50 per cent. of cases and mydriasis in from 20 to 30 per cent. Usually the pupillary change is bilateral unless an old specific iritis compels inequality and irregularity. Paralysis of the extrinsic muscles or of the muscles of accommodation is rare when there is myosis. Mydriasis is often observed concomitantly with atrophy of the papilla and diminution of visual acuity. Hence an amaurotic individual almost always shows mydriasis, which is the position of the iris in repose. There are, however, cases of tabes with myosis, even when there is optic atrophy. In about 30 per cent. of cases the pupils are unchanged.

Inequality of the pupils, or anisocoria, is almost as frequently present as myosis. The question to be answered is: Which is the abnormal pupil? Usually it is the smaller of the two, but the test is made by subjecting both to the action of the same amount of light and then noting which has the most vigorous response. If the pathologic pupil is mydriatic, it is by reason of irritation of the sympathetic or paralysis of the motor oculi. If it is the former, atropin will increase the mydriasis. If it is the latter, atropin will have no effect upon it. If the pathologic pupil is myotic, there is irritation of the motor oculi or paralysis of the sympathetic. If it is the former atropin will not affect it; if it is the latter, the pupil will still react to light or accommodation.

Irregularity of the pupil is especially common in mydriatic eyes. Often the pupil is elliptic, often it is caught down in one or more spots. The diagnostic value of this sign is considerable, if for no other reason than its precocity, since it usually antedates the Argyll-Robertson reflex. Atrophy of the iris is not infrequent. If one examines the iris with a pocket-lens, he will notice in the normal eye some bundles of fibers in relief, running in a radial manner next the pupillary opening, and in a circular direction in the front of the iris, next the sclerotic coat

When there is atrophy of the iris these folds in relief disappear in places or altogether.

Disturbances of accommodation are rare and unimportant, yet paralysis of accommodation occurs and may be complete or partial. Spasm of the muscles of accommodation may be present and cause not only imperfect focussing, but also periorbital pain, photophobia, and sometimes diminution of the visual field.

Of the disturbances of pupillary reflex action, the Argyll-Robertson sign is too well known to demand notice. According to the authors, this sign, found also in general paralysis of the insane, means syphilis. When present it is bilateral, but has been exceptionally found to be unilateral. Erb's sign consists in the loss of the pupillary reflex response to pain, no dilatation taking place when pain is produced. Gowers' sign consists in a reaction to light by oscillatory movements. Pilcz's sign consists in a narrowing of the pupils when the lids are closed, and their dilatation when the lids are opened. This sign has been regarded as occurring in normal patients, but Pilcz insists that it is far more common in tabes and general paralysis than in any other disease or in health.

Sensory disturbances. Deep sensibility to pressure of the globe is often abolished in tabes, there being ocular analgesia. Often, however, deep sensibility is unchanged, or there may even be hyperalgesia. The analgesia or hypalgesia may be unilateral or bilateral. Superficial sensibility in the tabetic eye may also be affected, anesthesia, hypesthesia, and hyperesthesia all being found. When these occur it is because of involvement of the trigeminal nerves, and they may be unilateral or bilateral. When once fully developed, the anesthesia is permanent. Subjectively, sensation may also be affected.

Trophic, vasomotor and secretory disturbances all occur. Trophic disturbances consist of corneal herpes and herpetic ulcerations, and neuroparalytic keratitis. When these occur there is nothing about them to render them distinctive. Vasomotor and secretory disturbances consist chiefly of increased flow of tears and epiphora, and occur commonly in the crises of the disease.

Most important is the atrophy of the papilla, occurring in from 10 to 30 per cent. of cases. It is an early symptom and may precede for years the other symptoms of the disease. It occurs between the ages of 30 and 50 years, generally in the preataxic stage. Ophthalmoscopic examination shows either a neuroretinitis or a simple or primary optic atrophy. Usually the onset is unilateral and deliberate, continuous, and gradually progressive. The first thing to disappear is the ability to distinguish shades of colors, there being dyschromatopsia. Usually green disappears first, then red, yellow, and finally blue. The contour of objects is not clearly seen, either, and false conclusions are drawn. Usually there is a progressive concentric narrowing of the visual field, but sometimes an irregular reduction is present. As a rule, sight as a whole is diminished and finally lost. When atrophy of the papilla is present, the case usually runs a reasonably benign course. Not a case seems ever to have been really cured, though some seem to have recovered spontaneously while certain remedies happened to be given.

Lightning Pains. Abadie and Nogue[1] allude to the fact that the lightning pains of tabes are usually regarded as constantly and early present in all cases of the disease. The lightning pain is distinguished by 3 characteristics: a sudden explosion, instantaneous disappearance, and great severity. Often these pains are the only symptom, and appear isolated or in groups or crises. They are usually located in the leg and lower part of the trunk, but may appear anywhere, even in the face. They are due to irritation of the posterior spinal roots or the sensory cranial roots.

From time to time cases turn up in clinics without lightning pains. Examination into the records of 400 cases in the service of Pitres has disclosed 16 cases (4 per cent.) in which, after painstaking inquiry, lightning pains were absent. Of the 16 cases, 8 had had tabes less than 5 years, and 6 more less than 10 years. When it is remembered that lightning-pains may appear late, it is apparent that some of these 16 patients may subsequently have them. Thus 4 per cent. is too high a percentage.

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(1) Jour. de Méd. de Bordeaux, Jan. 19, 1908.

Osseous and Arthritic Disturbances. J. Stefani[1] says that disturbances of bones and joints, far from being late manifestations of tabes dorsalis, very frequently appear exceedingly early. In support of his contention he reports 5 cases, the first at some length. In this case there were 3 fractures before signs of tabes appeared. The first fracture, in the upper end of the left tibia, occurred while the patient was simply walking along the street, without any fall or accident or trauma. She felt no pain when she dropped to the ground, but she could not rise. While still going about on crutches, she slipped, fell, and broke both the tibia and fibula about 7 or 8 centimeters above the malleolus. After recovering from this fracture she again broke her leg the very first day she attempted to go without her crutches. Yet up to the time of the last fracture the only suggestion of tabes was pupillary inequality, the right pupil being slightly dilated, with a hardly perceptible reduction in the reaction to light. There was no loss of the knee-jerk or the tricipital jerk, no lightning pains, no ataxia, no ocular paralysis, no sensory disturbances in the skin, though the first fracture was not painful. Yet the author feels sure that the patient was suffering from tabes. After reporting 4 other cases, Stefani divides up the cases of tabetic osteoarthropathy into 3 groups:

(1) The first form is purely osseous. It gives rise to 2 kinds of manifestations, spontaneous fractures of the bones, of frequent occurrence, and localized osteitis in the small bones of the extremities, of much less frequent occurrence. The pretabetic spontaneous fractures are in course exactly like those occurring late in the disease. Frequently multiple, they usually involve the femur, tibia, or fibula, more rarely the bones of the forearm. If due to trauma, the trauma is usually very insignificant and wholly incapable of causing the break. The fracture is slow in healing, the swelling and deformity are usually considerable; yet the break sometimes heals without much imperfection or deformity. The ultimate cause of these fractures is a rarefaction of the bones.

(2) The second form is the articular. In this form the onset of tabes is not by a fracture, but by an arthritis.

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(1) Gaz. des Hôp., Feb. 25, 1908.

The diagnosis is difficult and will be overlooked unless tabes is kept in mind. Gonorrhea may easily be ruled out by the absence of discharge, but various other hydrarthroses are not so easily differentiated. Sometimes the signs of tabes in these cases are very meager, Characteristic of tabetic arthropathy is an abrupt onset with acute inflammatory phenomena, accompanied only with a white, hard, edema, involving the periarticular tissue. As a rule careful search will show a trace of tabetic indications somewhere, though one of Stefani's cases here reported did not show such manifestations until after the development of the arthropathy.

(3) The third form, the more usual, is a combination of the first 2, constituting an osteoarthropathy. This often precedes the usual tabetic signs. The author says that "among the first signs capable of setting the clinician right as to the nature of these osteoarthropathies are the pupillary disturbances, the inequality of the pupils first, and the Argyll-Robertson pupil secondly. The absence of the knee-jerk is a comparatively late sign, as compared with the oculo-pupillary signs. Clearly the conclusion is justifiable that there are very early osteoarthropathies in tabes dorsalis, occurring even before tabes appears, and therefore deserving the appellation: pretabetic. The absence of all cardinal symptoms and signs of tabes in an osteoarthropathic case ought not to lead the clinician to conclude that tabes is absent, for it may subsequently develop.

Treatment. Milian[1] advocates the use of mercury in tabes. He believes it efficacious in preventing the development of tabes, if used freely during the secondary stage of syphilis. He gathers together the few and doubtful cases of recovery from tabes by the use of mercury, and infers that possibly cases may from time to time be so cured to-day. Of more value is mercury in causing certain symptoms of tabes to disappear, particularly the lightning-pains, though it is not always successful in aborting them. Finally it probably arrests many cases of tabes. [The Editor believes that the lightning pains of tabes disappear spontaneously when the nerve-fibers conducting the pain-impulses are destroyed, as they often ultimately are; and that many cases of tabes arrest themselves spontaneously is well known to all.]

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[1] Le Progrès Méd., June 20, 1908.

Debove[1] believes that tabes should be for the surgeon a *noli me tangere.* In support of this contention he instances cases of attempted resection of the knee which were anything but beneficial to the patient, his first case having lost a leg by it. Besides these cases, he brings forward others to show that gastroenterostomy is of little value in allaying gastric crises. Sometimes the operation is done by reason of a faulty diagnosis, as in a case where the crises were mistaken for pyloric stenosis. In other instances the gall-bladder has been operated on, and a floating kidney put in place in a vain attempt to cure gastric crises. Debove believes that as soon as the diagnosis of tabes is made the surgeon should leave the case alone, except under the most exceptional circumstances.

TABIFORM CHANGES IN DIABETES.

Changes in the posterior columns of Goll and Burdach entirely analogous to those occurring in tabes are reported by L. Schweiger[2] in diabetes, in 2 cases in Schlesinger's service. The changes found were not like those occurring in pernicious anemia, but consisted of a sclerosis in the column of Goll in the lumbar cord and the column of Burdach in the cervical region. There was in the territory involved a slight increase of neuroglia. At certain segments the zone of entering roots also showed the changes characteristic of tabes. In one of the cases the fibrous network in Clarke's column was somewhat reduced in volume. The only variation from tabes appeared to be quantitative rather than qualitative, there being many undegenerated fibers present among the degenerated ones. These changes seem to be quite independent of the multiple neuritis occurring frequently in diabetes, though they doubtless are both caused by the same toxic factor. A review of the literature shows 8 similar cases of diabetes with posterior sclerosis of the cord, and in only 2 of the 10 was either history or evidence of syphilis present, though, of course,

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(1) La Presse Méd., July 22, 1908.
(2) Wien. med. Woch., Aug. 3, 1907.

it possibly cannot be excluded. All of the 10 cases of diabetes were severe ones in which even starvation failed to get rid of the sugar; and in some of them there was also tuberculosis. These diabetic cases are closely analogous to those occurring in ergotism, and doubtless also to those produced by Spielmeyer in dogs by trypanosome injections.

SUBACUTE COMBINED CORD DEGENERATION.

Julius Grinker[1] reports 17 cases of subacute combined cord degeneration, a disease which has been described under many names, such as subacute combined sclerosis; subacute ataxic paralysis; combined cord degeneration; combined sclerosis of the Lichtheim-Putnam-Dana type; diffuse spinal cord degeneration; and the title which the author has chosen.

Symptoms. "The principal symptoms can be divided into (a) sensory symptoms, due to posterior tract degeneration, and (b) motor symptoms, consisting of weakness and spasticity in the early stages, due to pyramidal tract degeneration, which is followed in the terminal stage by flaccid paralysis, owing to supervening anterior horn degeneration." One of the earliest manifestations is paresthesia of the lower extremities, in the form of tingling, numbness, prickling or a dull ache. In other cases a sense of stiffness and fatigue appears in the legs. Ataxia soon follows. In exceptional cases the paresthesiæ are experienced elsewhere, as in the arms, or even in the tongue.

"The superficial reflexes do not show characteristic findings and the light reflex is usually normal, although a sluggish response to light has been reported in a few cases. The tendon reflexes are almost invariably found exaggerated quite early in the disease, and ankle clonus, Babinski's toe sign, Oppenheim's foot sign and Gordon's paradoxic reflex are common accompaniments of the pathologically exaggerated deep reflexes. Gradually the lower extremities become spastic and walking becomes very irksome because of incoördination and spasticity, two symptoms which have given the disease its name, 'spastic ataxia.' After a time, as the condition progresses, the patient is compelled to

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(1) Jour. Am. Med. Assoc., April 4, 1908.

take to his bed which he seldom leaves afterward, because of the successive development of complete paraplegia. There will usually now be found various degrees of anesthesia, analgesia, thermoanesthesia and loss of muscle and joint sense affecting principally the lower portion of the body. Such patients have difficulty in locating their lower extremities, except when they are aided by vision. Sphincter disturbances, if they occur at all, are usually slight in degree and may consist in nothing more than a desire to urinate frequently, or there may be a moderate degree of constipation or diarrhea.

"The cranial nerves, with the exception of the optic nerve, are practically never affected in this disease; optic atrophy is but rarely seen. Nystagmoid movements have been observed; this symptom probably resulted from extreme weakness of ocular muscles.

"Anemia is a common symptom in subacute combined cord degeneration; indeed, as has been stated, the first cases of this disease were invariably described in association with grave types of anemia. All varieties may occur, from the slight anemias ordinarily considered still within the range of normal to the most serious types of pernicious anemia, with high color index, nucleated reds and extreme poikilocytosis.

"After a period varying from 6 months to 6 years, the patient rapidly passes into the terminal stage: the deep reflexes become abolished and sphincter paralysis appears. This stage is of short duration. If the patient is not carried off by some intercurrent complication, such as pneumonia, decubitus, or cystitis, extreme emaciation with exhaustion usually closes the scene.

"The course of the disease varies from 1½ to 5 or more years, and is progressively downward. Prolonged remissions have been observed which occurred either spontaneously or were the result of treatment, but I know of no recoveries in this disease.

"The reported cases can be conveniently subdivided into 3 groups: 1. Those in which pernicious anemia was present from the beginning. 2. Cases in which a grave type of anemia, not necessarily of the pernicious variety, was at some time associated with the disease and constituted an important sign. 3. Cases in which the anemia, if present at all, was insignificant and constituted a minor symptom of the entire disease. Cases of the first group have been frequently described, and are therefore best known." The second and particularly the third group of cases, though relatively more common, are less often recognized than the cases of the first group.

The case histories of individuals from each group are recounted. "In the entire series there were 14 males and only 3 females. Nearly all were over 40 years old. In only 1 case was there a history of past syphilis. Nearly all began with subjective sensory disturbances and disorders of coördination and were followed by motor weakness. In several cases gastrointestinal disorders, and particularly diarrheas, appeared either before the beginning or during the course of the disease." Only 4 cases began as a typical pernicious anemia, while the others either showed but little anemia or the anemia was not an early symptom.

Differential diagnosis. Early in its course the disease may be mistaken for tabes, because of the sensory disturbances common to both affections, but in tabes the lancinating pains, Argyll-Robertson pupil and visceral crises are prominent early symptoms not found in combined cord degeneration, while muscular weakness, exaggerated reflexes, and Babinski's toe sign occur reasonably early in this disease and are absent in tabes. In the later stages the two diseases cannot be confused. Spinal syphilis and multiple sclerosis offer great difficulty in the differentiation from combined cord degeneration. Multiple sclerosis is usually differentiated by a careful sensory test.

Prognosis and treatment. "The disease usually terminates fatally with 1½ to 5 or even 6 years. Instances of partial and even complete recovery have been recorded, but sufficient latitude must be allowed for mistakes in diagnosis. There is no valid reason, however, why improvement and even arrest of the disease should not occur if treatment is begun sufficiently early.

"As the cord disease is frequently found in company with anemia or some other form of ill health, the associated disorder will come in for a large share of attention. At all events, rest of mind and body is positively indicated. If anemia is present, iron tonics and arsenic, particularly the latter, are indicated. If gastrointestinal disorders, such as indigestion or diarrheas, are troublesome, strict attention will be paid to the dietary and appropriate remedies administered.

"The sensory discomforts, which are usually only subjective in the beginning, can be greatly benefited by the application of the faradic brush. The Pacquelin cautery applied over the spine occasionally relieves sensory symptoms. General faradization as a tonic and local faradization over weak muscles, used in moderate strength and not longer than 20 minutes at each sitting, appear to be beneficial; in addition, gentle massage may be used cautiously. Patients must be warned against every form of fatigue; they are advised to lead a regular life and the early cases are permitted to indulge in a moderate degree of open air exercise.

"For the incoördination nothing has yet excelled or even approached in efficacy Frenkel's systematic exercises originally devised for the treatment of tabetic ataxia. These can be varied to suit each case and will be found very helpful in the early and even in the more advanced degrees of incoördination encountered in this disease.

"It is needless to say that there is no specific remedy against this disease. The treatment must, therefore, be purely symptomatic. Bearing in mind our utter helplessness in the face of the fully developed disease, it will be our endeavor to recognize the disease in its early stages when treatment can accomplish the most good."

Clinical Case. Degeneration of the spinal cord, associated with severe anemia in a case of chronic gastric ulcer, is reported by T. K. Monro and M. B. Hannay.[1] Reference is made to the early cases of such degeneration, as reported by Leichtenstern, Lichtheim, and others, and attention is called to the fact that the anemia under such circumstances did not always present the features which might be regarded as characteristic of pernicious anemia; furthermore, that the anemia might appear before, along with, or after the onset of the nervous symptoms, or that there might be no anemia at all. In these cases it would seem at times that some toxin must be responsible for both the anemia and the changes in the cord, as suggested

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(1) Glasgow Med. Jour., February, 1908.

by Russell, Batten, and Collier.[1] At any rate these authors have shown that the degenerated tracts correspond exactly to the area of distribution of the peripheral arteries derived from the vessels of the pia mater. The present case occurred in a young man, 28 years of age, a compositor, who, a year before admission, had rheumatic pains in the knees off and on for 3 months, then an intermission in his illness, followed later by stiffness in the feet and legs, and some ataxia. Pain in the stomach developed later in the case and signs of ulcer of the stomach with marked anemia. The number of erythrocytes sank to 2,500,000 and the hemoglobin to 30 per cent. Examination on admission showed him to be languid and thin, responding slowly and deliberately to questions, his skin and mucous membranes pale; there being no cough, pain, abnormality of ocular muscles or tremor in the hands or face, the muscles being firm. The knee-jerks were lost, the plantar reflexes were doubtful but mainly flexor, there was incoördination in both legs, especially the left, the gait was stiff and slow, with a wide base, and there was staggering on turning. There was no loss of power in the legs or toes, and no loss of sensation.

A postmortem examination revealed the gastric ulcer, three-quarters of an inch by half an inch, on the lesser curvature, and changes in the cord, chiefly of well marked sclerosis of the posterior columns, especially in the lumbar region. There was also marked sclerosis of the posterior roots and of the tract of Lissauer. The vessels in all the sections examined were normal in appearance.

"The present case does not correspond to the type established by Russell, Batten, and Collier, either from the clinical or from the anatomical point of view. No doubt there was the ataxy or ataxic paraplegia of the first stage, but those evidences of profound and progressive changes in the spinal cord, which characterize the second and third stages of subacute combined degeneration, did not show themselves here. Neithet did anatomical investigation reveal a diffuse focal lesion of the white matter in the dorsal region, with secondary ascending and descending degeneration of the long tracts above and below. On the contrary,

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(1) **Brain, xxiii, pages 39-110.**

the changes in the cord were suggestive of a primary neurone degeneration like that of tabes, with its intensity greatest in the lumbar region, and with the addition of a very slight degeneration in the pyramidal tracts.

"It is reasonable to suppose that the toxin which induced the degeneration in the cord, and which was probably also the main cause of the anemia, was evolved, or at any rate absorbed, at the seat of ulceration in the stomach. It may be remarked that the occurrence of system degeneration in the spinal cord is well known in connection with other morbid blood states besides anemia, since it may be met with in diabetes, and in certain forms of grain poisoning, such as ergotism, lathyrism, and pellagra."

ACUTE MYELITIS.

Optic Neuromyelitis. Under the caption "Optic Neuromyelitis." Lannois and Gauthier report a case of acute myelitis with optic neuritis.[1] Knowledge that disturbances in the fundus of the eye are very common in chronic spinal maladies is widespread, but it is not generally known that optic neuritis and acute myelitis may develop concomitantly. Devic in 1894 collected a few instances of this combination, and proposed the title "acute optic neuromyelitis." It is a case of this sort that the authors present. Hillion, in his *Thèse de Paris* in 1907, was able to collect from the literature only 45 of these cases. Briefly summarized, the case occurred in a girl, 18 years of age, who in May, 1907, was seized with a complete paraplegia, abolition of the tendon-reflexes, anesthesia below a point about 20 centimeters above the groin, retention of urine, and constipation. About three weeks later the paralysis ascended. First there was a paresis, and then a complete paralysis of the right arm, paresis of the tongue, and disturbances of phonation, and right facial paralysis. Later a sacral bed-sore developed. Lastly, about a month after the onset of the disease, the optic neuritis appeared in the left eye. At this time, her condition which had been very grave, began to improve, and the paralysis to disappear. But three months later, when the sight had begun

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(1) Lyon Médicale,, May 10, 1908.

to reappear in the left eye, the right eye became involved. The sight of the left eye was then again impaired and finally lost. Ophthalmoscopic examination showed first an edematous papillitis of the left eye, then of the right, and next a complete atrophy of the papilla in the left eye, and a papilla simply a little paled in the right. After the fourth or fifth month, improvement again set in, and was evidenced by the cerebrospinal fluid, the lymphocytosis found during the acute stage of the disease having almost completely disappeared.

The *symptomatology* of this affection or syndrome presents nothing unusual beyond the simultaneous and concomitant development of the two lesions, acute myelitis, and optic neuritis. Nevertheless the above case presents certain points which the authors emphasize. In the first place there is the onset of the myelitis with paraplegia. This mode of onset is much less frequent than other modes, for in Hillion's 45 cases, only 10 showed onset with paraplegia. In fact, most cases began with visual disturbances, foggy and blurred vision, and then a progressive loss of sight, leading rapidly to a total amaurosis. The spinal symptoms followed later.

Moreover, the rule is that both eyes are equally although not strictly synchronously involved. Usually only a few hours or days intervene between the appearance of the optic neuritis in the two eyes, whereas in this case the interval was 3 months. In only 2 other cases has so long an interval been recorded. A third point is that this case ran a subacute rather than an acute course.

As to prognosis, two symptoms denote a grave situation: the appearance of a sacral decubital sore, every case of which has terminated fatally, and an ascending paralysis. In 12 cases in which ascending paralysis was noted, 11 terminated in death. Yet in the present case death has not followed, though both of these grave symptoms were present. The etiology of this and all cases is obscure.

The authors recall another case reported in 1894 as an example of multiple sclerosis. They now believe it to be a case of optic neuromyelitis, because the postmortem findings disclosed the coexistence of lesions of the motor tract and of the optic nerves.

ACUTE HEMORRHAGIC MENINGOMYELITIS.

A case of acute hemorrhagic meningomyelitis with secondary softening and cavity formation, and having some bearing on spinal localization, is reported by J. H. Lloyd.[1] The patient, a male, colored, 34 years of age, with a history of syphilis, was taken ill with numbness in the legs and some loss of power. This kept growing worse, so that in less than a week he was paralyzed in both legs. He also had a girdle pain about his waist, and lost entire control of his bowels and bladder. He was burned on each foot by hot bottles, which he did not feel. A deep bed-sore formed over the sacrum. All of the above symptoms developed within 5 weeks' time. At his admission there was complete paraplegia, but no contractures. The knee-jerks were absent, which is noteworthy. The abdominal, cremasteric, and plantar reflexes were gone. Below a level drawn around his waist at a little below the lower margins of the ribs, and 2 inches above the umbilicus, there was total abolition of sensation of every sort. Lumbar puncture gave 15 c.c. of clear fluid, acellular and sterile. The patient ran a febrile course, with one peculiar phenomenon: sweating above the line of anesthesia, and a dry skin below it. Death took place just 9 weeks after the numbness began in the legs.

The cord was examined with great care, and numerous sections were made. "Summarized, a study of these sections showed that below the fourth lumbar segment there was no lesion. Beginning in the fourth lumbar segment and extending into the eleventh thoracic segment, there was an area of intense meningomyelitis, with the following characteristics: The pia, especially the posterior portion, was much thickened, apparently by a fibrinous exudate rather than by proliferation of the tissues. There was an intense infiltration of cells, leucocytes and lymphocytes, about the blood vessels in the pia. The pial vessels were full of blood; all the walls of the smaller were thickened. The walls of the larger blood vessels did not show any proliferation of the intima. The posterior roots as they entered the cord were apparently constricted by the swollen meninges, and just outside of the meninges the

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(1) Jour. Am. Med. Assoc., Dec. 7, 1907.

nerve roots were swollen and the nerve fibers reduced to a granular débris. Not all the posterior roots were affected in this way; some were normal. Within the cord the blood vessels were congested, and there were numerous hemorrhages, especially in the gray matter; these were of recent occurrence, as shown by the partial preservation of the form and color of the red blood corpuscles.

"The idea that the case was of syphilitic origin is borne out by the history of syphilis, by the exudative leptomeningitis, by the thickened arteries, and by the acute hemorrhagic myelitis with secondary softening and cavity formation.

"The localization in this case pointed to the eighth thoracic segment. The sections showed that the cavity ended in that segment rather abruptly and that there was only slight evidence of degeneration in the seventh segment. Sherrington has shown in his experiments on monkeys and cats that there are what he calls 'overlaps' in each area of skin innervated by the dorsal nerve roots. Thus the band-like area innervated by the eighth segment, for instance, has also sensory filaments from the seventh above and from the ninth below. In other words, there is no area supplied exclusively by any one segment (as Türck and others of the older physiologists taught), but every thoracic area is supplied by at least three segments, viz.: its own segment and the one above and one below. According to this view, a lesion located in the eighth thoracic segment would cause absolute anesthesia only in the lower half of its own skin area and impaired sensation in the upper half; this because the upper half is supplied from the seventh segment in part. If this holds true in this case, it tends to put the area supplied by the eighth segment in about the same location as given in the diagram of Head, who places the umbilicus at the lower margin of the ninth area. It also corresponds closely to the mappings out by Sherrington on the body of one of his monkeys."

ACUTE POLIOMYELITIS

Pathology. Very extensive work on the pathology of acute poliomyelitis and allied diseases has been done by F. Harbitz and O. Scheel, of the Pathological Institute of the University of Christiania. A lengthy article in Norwegian is made the basis of a condensed paper in German.[1] These authors studied altogether the cords, brains, and nerves of 17 individuals, 9 being males and 8 females. Ten were children from 9 months of age to 11 years; 7 were adults from 18 to 38 years. In 13 cases death took place in the acute stage from 2 to 10 days after the onset; in most of these cases death occurred from the seventh to the ninth day. The cord was examined by serial sections made at from 6 to 8 levels, in some cases even more. The cord membranes were also studied. The medulla, pons, and adjacent part of the cerebellum were in each case studied *in toto* in frontal sections, some 8 or 10 sections being studied in each case. Other frontal sections were made, passing through different parts of the great central ganglia of the brain, including the *basis cerebri.* Still other sections were made through the *centrum ovale* and through large portions of the cerebral cortex, especially about the fissure of Sylvius. All these sections, including the membranes where possible, and in some cases the dura, at least 50 in number for each of the 17 cases, were carefully studied. In addition, several different peripheral nerves and spinal ganglia were examined. Staining by eosin and toluidin blue gave the best results.

Microscopically the meninges always showed a meningitis, though no inflammation was visible macroscopically. The pia was always found infiltrateed with cells resembling lymphocytes, and was especially involved in the lumbar portion. This infiltration was always about the vessels. [See the article of E. Farquhar Buzzard, Practical Medicine Series, 1907, Vol. X, p. 143.] The inflammation of the gray matter of the cord consisted of not only a hyperemia and edematous exudate, but also of a cellular infiltration spreading from the blood-vessel-wall. The infiltrating cells were lymphocytes, plasma-cells, and cells resembling Maximaw's polyblasts, but most of them were ordinary polymorphonuclear cells. These cells proliferate at the apparent expense of the ganglia cells. The distribution of the inflammatory process in the cord was about the same in all cases: the process was most pronounced in the an-

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(1) Deut. med. Woch., Nov. 28, 1907.

terior horns, less so in the posterior; and occasionally Clarke's vesicular column and the central gray matter, ordinarily free, were involved. The whole cord was involved from conus terminalis to medulla, the lumbar portion as a rule suffering most, the cervical enlargement next.

The medulla and pons and basal parts of the brain, in all of the acute cases except one, showed an inflammation starting from the vessels of the pia and spreading into the nerve-tissue. Without going into all of the very interesting details of the paper, it is enough to say that acute poliomyelitis is an inflammation which in lethal cases extends over the whole length of the spinal cord, medulla, and pons, and over large parts of the brain. There is always a meningitis, which is the primary site of the inflammation. This follows the vessels from the pia into the brain and cord, which are invaded therefore from the walls of the arteries supplying it with blood. The clinical course of the case seems to be determined by the preponderance of the inflammation in any given region. The term acute poliomyelitis may perhaps be retained, but surely acute bulbar paralysis, meningo encephalitis, and probably some cases of transverse myelitis, belong to this type. There seems to be no connection, however, between acute poliomyelitis and multiple neuritis, for in no one of the 17 cases were changes found in the nerves.

Very interesting is the comment of these authors upon the relation of acute poliomyelitis to Landry's disease. They say very properly that cases of acute poliomyelitis in children, with ascending symptoms, resulting fatally, are called acute poliomyelitis; but that cases occurring in adults with ascending symptoms are called cases of Landry's paralysis. Yet pathologically acute poliomyelitis of adults and Landry's disease are the same things. So also the polioencephalitis inferior [et superior] acuta, is nothing more than acute poliomyelitis, with lesions predominating in the upper part of the cerebrospinal axis.

The epidemic appearance of acute poliomyelitis in Norway from 1903 to 1906 proves it to be an infectious disease with a living virus, and possibly it is a diplococcus which is the infectious agent. [See Practical Medicine Series, 1907, Vol. X, p. 145.] The pathway of the infection along the course of the arteries leads one to the conviction of a primary injection of the pia and a rapid spread to the brain and cord tissue. In the cord the infiltration is especially intense in the anterior middle fissure, where many arterial twigs pass into the cord from the anterior spinal artery. Thus the anterior horns are most and primarily involved, since the supply of the branches of the anterior spinal artery is to these horns. When the pia is free the underlying vessels and brain are free from inflammation; and where the pia is involved the brain or cord and vessels beneath it are involved. What is true of the cord is true of medulla and pons, most of the arteries entering on the anterior surface, which is the most infiltrated. In general, however, the inflammation here is more in spots than in the cord.[1]

Symptomatology. Byrom Bramwell[2] presents an analysis of 76 cases of poliomyelitis anterior acuta. As to sex, 33, or 43.4 per cent., of the cases were males. As to age, 16 occurred during the first year of life, 8 during the second year, 17 during the third year, and 7, 6, and 5 respectively during the 3 succeeding years of age. One case each was reported in the sixteenth, eighteenth, twentieth, twenty-fourth, and thirty-fifth year of life. Of 73 cases in which the age is stated, 41, or 56.1 per cent., occurred in the first 3 years of life, and 59 cases, or 80.8 per cent., in the first 6 years of life. As to etiology, 51 of the 76 cases developed apparently without cause, most of the patients being previously in good or robust health. In the other 25 cases all sorts of illnesses and accidents were alleged, from measles and pneumonia to fracture of the radius and a long walk.

As to the *initial symptoms,* a good history was obtained in only 47 cases. In 46 of these fever occurred early, vomiting was early present in 13, headache in 10, convulsions in 6, coma in 2, and retraction of the head in 2. In 14 of the 76 cases pain was stated to have been present during the initial period, being absent in the remaining 62 cases, or not being noted. The pain occurred almost anywhere, in the back of the neck, between the shoulders, in the back,

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(1) The article of Harbitz and Scheel was translated and published in the Journal of the American Medical Association, October 26, 1907.

(2) Scottish Med. and Surg. Jour., June, 1908.

in the legs or arms, and in the knee. Pain persisted after the initial symptoms had passed off in 5 cases, and lasted even as long as 6 weeks. In 1 case there was persistent paralysis of the bladder, which lasted several weeks, and in another case both the bladder and the rectum remained paralyzed for 5 months after the initial symptoms passed off.

The paralysis was of sudden onset, being the first and only symptom in 1 case. In 9 cases it developed in the first day of illness, in 8 cases on the second day, in 3 on the third day, in 3 on the fourth day, in 2 on the fifth day, in 1 on the sixth day, in 3 cases within a week, and in 1 case each 2, 4, and 6 weeks after the onset of fever. These are the only cases of the 76 on which data covering this point were recorded.

The distribution of the paralysis was as follows: both legs, 18 cases; right leg, 12 cases; left leg, 17 cases; right arm, 6 cases; left arm, 5 cases; all four limbs, 6 cases; all four limbs and trunk, 4 cases; right arm and right leg, 3 cases; right arm and left leg, 1 case; right arm and left sternomastoid and trapezius, 1 case; both legs and right arm, 1 case; both legs and left arm, 2 cases.

A residual paralysis remained in 75 of the 76 cases, involving in order of frequency the left leg, right leg, and both legs. In general, paralysis of the upper extremities showed a much less tendency to be permanent than did paralysis of the lower extremities.

The New York Epidemic. Last year an epidemic of acute anterior poliomyelitis prevailed in New York, beginning in August, 1907. The following table, taken from an article by Joseph Collins,[1] shows its proportions, the figures being merely for those cases which appeared at the Hospital for the Ruptured and Crippled in New York:

		Anterior Poliomyelitis.
October,	1906	44 cases
November,	"	27 "
December,	"	16 "
January,	1907	19 "
February,	"	18 "

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(1) Med. Record, Nov. 2, 1907.

March,	"	25	"
April,	"	26	"
May,	"	21	"
June,	"	27	"
July,	"	55	"
August,	"	116	"
September,	"	156	"
		550	cases
October 1-15, 1907		57	"
		607	cases

Thus in August and September, in 2 months, there were 272 cases, as against 278 for the previous 10 months. There are no means of finding out how many cases have existed in New York, but Collins thinks it safe to infer that not more than one-quarter of the cases have come to the hospital. This would indicate that there have been upward of 1,000 cases.

"The noteworthy symptomatic feature of the present epidemic is the considerable percentage of cases which make a fairly good recovery, *i. e.,* a recovery without marked atrophy, and in this respect I think the epidemic now prevalent differs from what has heretofore been described. In marked contradistinction to this is the relatively large number of cases in which the oblongata and the pons have been the seat of the lesion. Naturally, the majority of such cases do not come to the dispensary, because they succumb in a comparatively short time after the occurrence of the disease."

"As to the cases that terminate in recovery. In all the epidemics, so far as I have examined the literature, there have been recorded recoveries, but they are very few, and the idea of complete recovery from what seems to be a genuine, fully developed case of anterior poliomyelitis, was to me quite novel. It is too early to give statistics of the cases that have recovered. Many of them are still under observation. Some of them will not eventuate in complete recovery. We consider a case recovered that does not exhibit muscular atrophy. The kind of cases that we are seeing in our clinics have a history somewhat as follows: The child sickens with the ordinary symptoms of the disease, and in customary way develops paralysis of one or more extremities. After two or three weeks, in some instances a shorter period, the child begins to move the paralyzed members, and the recovery progresses until he is able to use them all, save perhaps one or more groups of muscles. So far the occurrences are as they are related in the books. But in the cases of which I speak the groups of muscles which have not up to this time regained their function, do not begin to atrophy, as they do in the ordinary case; they gradually regain their function."

Henry W. Berg[1] discusses the same epidemic, his article being based upon an experience with 25 cases. "The clinical manifestations of the disease were in some cases those classically recognized as infantile spinal paralysis. In many of the cases, however, the affection manifested itself as a combination of the symptoms of anterior poliomyelitis and those of acute bulbar paralysis, giving a picture which might well be termed pathologically poliomyeloencephalitis, although such a term would be objectionable in that it indicates an involvement not of limited portions of the cord or brain, but a general inflammation of the cerebrospinal axis. To this group characterized by bulbar paralysis, in addition to the paralysis of the extremities, three of my cases belonged, and all of these died. The remaining cases were ordinary cases, corresponding in a general way to classical cases of poliomyelitis anterior, for the most part divisible into two groups; that is to say, cases which I saw less than seventy-two hours from the onset in which the fever was still present, while there was an extensive paralysis, and cases seen equally early in the history of the disease in which the paralysis was more or less extensive, with no fever present at the time of my examination, and only a history of fever less than two days in duration.

"It has been accepted that, while in the sporadic cases the extreme paralysis of the stage of onset undergoes regression, hence the term regressive paralysis (Barlow), no case absolutely recovers with disappearance of all paralysis. In all cases a more or less extensive paralysis remains for life. On the other hand, in this epidemic, in my limited

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(1) Med. Record, Jan. 4, 1908.

experience—for this epidemic must have numbered 2,000 cases in this section of the country—several times my prognosis as to the permanent nature of a part of the paralysis has not been confirmed. Two of the cases recovered entirely within six weeks, no obvious paralysis remaining."

"One peculiarity that stamps the epidemic form of the disease and distinguishes it from the sporadic form, is the fact that it occurs in older children as well as in infants. Indeed, young adults have not been free from it. Children from 5 to 8 years of age were not infrequently affected by it. This is a very important distinction between the epidemic and sporadic disease, which owed its name, acute infantile spinal paralysis, to the fact that the sporadic form of the disease was not known to affect adults.

"The symptomatology of the epidemic type of this disease is very different in many respects from that of the acute stage in a sporadic case. The fever is of greater duration in the epidemic cases, it is a more prominent factor, and is never absent in any of the cases, while sporadic cases frequently occur in which a healthy child has gone to bed at night and waked in the morning with the characteristic spinal paralysis. Many of the children were delirious and semicomatose, a condition from which they emerged very slowly. There was no vomiting except at the onset. There was hyperesthesia of all the surface of the body, and in many cases seen on the first, second, or third day there was some rigidity of the neck and spine. Even the paralyzed limbs could not be handled without pain. The patella reflexes were, in most of the cases, absent. The initial rigidity at the back of the neck soon gave way to a flaccidity of the muscles about the neck, in some cases, particularly those in which the muscles of the back were paralyzed. Three of my cases had bulbar symptoms and died of symptoms due to the involvement of the hypoglossal, glossopharyngeal, and pneumogastric nuclei. One of these cases had a comparatively low temperature, mental condition absolutely clear, and yet total paralysis of all the extremities and muscles of the back, together with the muscles of deglutition and speech, and finally died of diaphragmatic and cardiac paralysis. In one or two of the cases of this epidemic there was a temporary, at least, analgesia (pin test) in some of the paralyzed muscles, from which the patient recovered about the time when the fever disappeared, so that clinically in these epidemic cases symptoms are present not met in the sporadic cases of the disease, presupposing for their explanation a general inflammatory disease of the cerebrospinal axis, including nuclei in the pons and medulla, and longitudinally a more or less extensive myelitis, not only of the anterior gray horns, but of the posterior gray horns, the white columns, the anterior and posterior nerve roots, and even the meninges.

"It appears, then, from what has been said concerning the differences between sporadic poliomyelitis anterior and so-called epidemic poliomyelitis anterior, or, rather, as I think it should be termed, epidemic meningomyeloencephalitis, that the two conditions are radically different diseases, the latter including among its extensive neurological lesions the pathological changes characteristic of sporadic poliomyelitis. The clinical manifestations of the two diseases differ as markedly as is necessitated by the widely different pathological changes characteristic of each. The two conditions, moreover, differ in their morbidity, in the ages of the patients for which each of the conditions has a predisposition, the sporadic disease being found in children almost exclusively, the epidemic disease affecting adults and children. They probably differ, moreover, in a very important characteristic, for sporadic poliomyelitis anterior cannot be considered an infectious disease, while epidemic meningomyeloencephalitis may be, although positive proof of its infectious nature is still lacking."

LANDRY'S PARALYSIS.

Course. A case of Landry's paralysis with recovery, partial relapse, and then complete recovery, is reported by J. K. Mitchell.[1] "There is certainly a symptom group which cannot be put under the heads of multiple neuritis, of spreading myelitis, or of poliomyelitis, but which does fit the description of 'acute ascending paralysis' or Landry's disease. The points of diagnostic distinction are that the onset should be acute, its character generally ascending, with paralysis more or less complete, but with

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(1) Jour. Am. Med. Assoc., Feb. 1, 1908.

little or no change in sensibility, entire loss of the deep reflexes, and usually of the superficial ones, without involvement of the sphincters, and with no distinct or at least no characteristic pain. One of the stumbling-blocks of those anxious to settle the classification and pathologic relations of the disease has always been the innumerable supposed causal troubles described: syphilis, alcohol, infected wounds, cold, exposure, and so on.

"The condition is probably due to some toxin produced by organisms, unknown or unidentified, present in the blood or the tissues, this toxin product having a selective action on the spinal cord.

"Multiple neuritis offers in its symptom-complex the nearest approach to the symptoms of Landry's disease. Its onset is rapid, degeneration reaction is early present, the reflexes are lost; but the trunk muscles are not affected in polyneuritis; there are marked sensory changes, pain and tenderness of high degree. The paralysis, instead of ascending, either appears almost simultaneously in all four extremities or attacks the limbs in pairs, and muscular atrophy shows itself very quickly, usually becoming extreme in some muscles. The spread of the affection in polyneuritis, while sometimes very rapid, is less even and symmetrical in its advance. It is rare to find distinct signs of involvement of the cord in pure neuritis, however severe the neural inflammation may be.

"The patient had been exposed in a cold rainstorm five or six days previous, was thoroughly wet and chilled, and was obliged to continue at work. The next day he felt rather miserable, but went to work as usual. In walking to work he felt heavy and his use of his legs was awkward. An hour later he found himself unable to lift packages and heavy boxes, which was part of his duty. Before noon he was unable to walk except with support, and when he attempted to return home gave out before reaching there and had to be carried. He grew worse rapidly.

"On examination on April 13 he had complete paralysis of all four extremities. The hands could be moved a little, but the arms could not be raised from the body, the only remaining movements of the limbs being partial flexion of the forearms. The legs were totally immovable, though one or two muscles seemed to contract in the legs

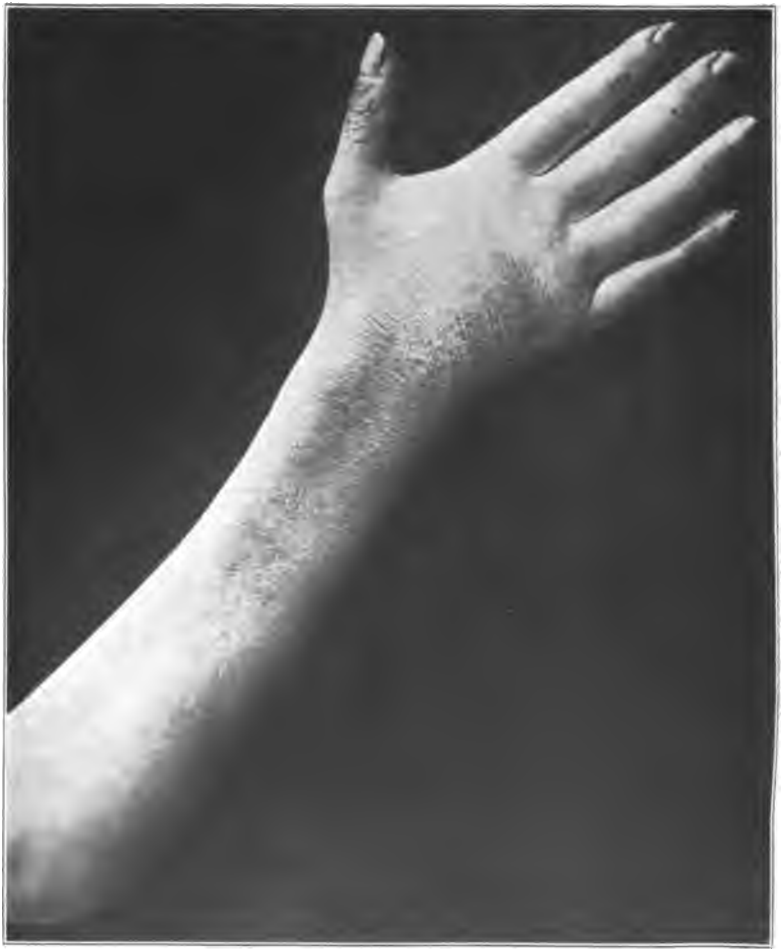

PLATE VI.

Sensory and trophic neuritis following herpes.—F. Rose (page 200).

to voluntary effort, a trifle more actively in the left leg than in the right.

"*Treatment.* The patient was ordered galvanism to the spine, stimulating massage, alternating applications of hot and cold water to the back, and small doses of ergot. Eleven days after his admission the cremaster reflex reappeared distinctly on the left side. He was able to make a slight attempt at movement of the left foot in extension. No wasting was observed, nor did he seem to have any definite loss of general muscle substance. The reactions of degeneration were at this time still more marked in the areas already affected.

"Sensation was again examined and showed normal condition. The only added observation of these first ten days was that the breathing apparatus seemed very slightly involved during the first week, but this difficulty did not increase and improved with the other symptoms from May 24. By the end of a month from the time of his admission he could turn over in bed, flex the legs on the thighs and the thighs on the abdomen, if the weight of the legs were supported. The power of extension was not improved. He could flex the forearms, but grip in the hands was almost completely absent. There was still no atrophy." By July he was walking well. In October he had a slight relapse, but since then he has entirely recovered.

A case of acute ascending paralysis with recovery is reported by C. W. Vining.[1] The patient was a well-nourished man, aged 24 years. His illness began with a feeling of weakness in the calves of his legs, quickly followed by blurring of vision, paraplegia, and difficulty in articulating. "On admission he was unable to walk, but could just stand with assistance. There was also considerable weakness in the muscles of his arms, especially in the extensors. The muscles were flaccid, but not wasted, and there was an absence of all reflexes, including the palatal reflex. The respiratory, abdominal, and intercostal movements were normal. There was slight right-sided facial weakness. The external muscles of the eye were normal. The right pupil reacted sluggishly to light and to accommodation, and was somewhat larger than the left. The

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(1) The Lancet, Feb. 8, 1908.

fundi were normal. Speech was a little indistinct, apparently due to difficulty in the movements of the tongue, and there was slight difficulty in swallowing. There was no alteration in the sensory functions except that he appeared to have lost sensation of the palate. His mouth was dirty with sordes on the lips. A lumbar puncture was performed soon after admission and 15 c.c. of clear fluid with a few whitish floating particles were obtained. In this an organism having the appearance of a streptococcus was found, but no growth could be obtained on culture media." A culture made from the throat was negative. Subsequently the muscular weakness became excessive, and the diaphragm was involved, reducing chest expansion to a minimum. Marked cyanosis and dysphagia followed, together with incontinence of urine and feces. Oxygen and strychnin were resorted to. Later some muscular wasting appeared, and there were some slight, rather indefinite alterations in sensation over the legs, especially the right one, with severe pain in the knees. Subsequently the diaphragm began to be used again, and all the paralyses cleared up, leaving no residual paralysis, although muscular wasting of the limbs was marked and the knee-jerks remained absent. The electrical reactions taken during the illness showed no reaction of degeneration, but there were reduced responses to faradism and no response to galvanism.

The interest of this case consisted in the recovery from what appeared to be a severe example of Landry's paralysis and which when the diaphragm showed signs of paralysis threatened to be speedily fatal. The diagnosis lay between (1) diphtheritic paralysis, (2) Landry's paralysis, and (3) a toxic paralysis affecting the anterior cornua or nerves. There was, however, no evidence or history either of diphtheria or of any exposure to such toxic influences as syphilis, gonorrhea, lead, or alcohol, and on the whole the case seemed to correspond better with Landry's acute ascending paralysis than with any other disease. Against this diagnosis there may be urged (1) the anesthesia of the palate and the slight sensory disturbance of the legs, and (2) the fact that the parts affected were not altogether anatomically in succession from below upwards, but in Landry's own description of the condition diminution in cutaneous sensibility over the distal parts of the limbs is mentioned, and the arms and face were affected before the legs were completely paralyzed. [It is suggested that there is nothing in the case to rule out the possibility of acute poliomyelitis of adults.—Ed.]

A case presumably of Landry's paralysis is recorded by Arthur Münzer.[1] The patient was received Sept. 1, 1907, with the statement that three days before he was suddenly seized with headache and malaise. The next day paralysis appeared in both legs. On the third day both arms were affected, and shortly thereafter speech and swallowing became difficult. At entrance there was pronounced dyspnea, moderate cyanosis, difficulty of speech, paralysis of the palatal muscles and of the muscles controlling swallowing, absent reflexes, flaccid quadriplegia, no fever, the pulse regular and not accelerated. A diagnosis of Landry's paralysis with fatal prognosis was made. A postmortem examination was secured and showed in the spinal canal an extensive hyperemia of the epidural tissue. In the cervical cord the meninges were deeply injected, and where the arachnopia was reflected upon the intradural nerves the same injection was found. The cord was soft, the gray substance very much reddened without visible hemorrhages, the white substance being apparently uninvolved. The base of the brain was also strongly infected, and there was a moderate hyperemia of the brain without other essential change. The intestines showed morbid inflammation. Microscopically the cord showed an exquisite poliomyelitis acutissima, especially in the lumbar portion. The bloodvessels of the gray matter were surrounded by an infiltrate of round cells, and evidently the vessels carried the substance injurious to the cord, this toxic material being unmistakably derived from the intestines. This case was undoubtedly the result of a gastrointestinal intoxication. This case Münzer regards as confirmatory of Harbitz's assertion that many cases of Landry's paralysis are cases of acute poliomyelitis with bulbar symptoms.

Carl D. Camp[2] reports a case of acute unilateral ascending paralysis, in a laborer, 60 years of age. He was admitted to the hospital April 6 and showed paresis of the left upper extremity, very slight in amount. The biceps

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(1) Berl. klin. Woch., June 29, 1908.
(2) Jour. Am. Med. Assoc., Nov. 30, 1907.

and triceps jerks were slightly increased on the left side. Sensation was everywhere normal. The left leg was almost completely paralyzed, but there was no Babinski toe-sign. Weakness in the left upper extremity gradually increased, so that in six days the left hand and arm were cold, stiff, and apparently in a beginning contracture. The left leg was now in a state of flaccid paralysis. On April 14 he was stuporous, and in showing his teeth, the left side of the face was seen to be slightly paretic. He died April 16. The postmortem was made within 18 hours. The small blood-vessels in the posterior columns of the spinal cord were especially prominent because of their thickened walls. "In the lumbar region the pia was slightly thickened and there was slight infiltration about some of the blood-vessels within the spinal cord. The blood-vessels were moderately sclerosed." [These conditions remind one of the investigations of Harbetz and Scheel, page —, and suggest the possibility of the above case being really a case of unilateral acute poliomyelitis of the adult. Possibly, because of the preliminary exaggeration of the reflexes, Camp would rule this out, despite the postmortem findings.—Ed.]

DISEASES OF THE PERIPHERAL NERVES.

Multiple Neuritis.

Etiology. W. Poljakoff and W. Choroschko[1] report a case of multiple neuritis due to the *Bacterium coli*. The patient, a young woman, 25 years of age, was taken ill with some acute infection. From the bladder urine was taken which was found to contain the bacterium coli. Signs of multiple neuritis, rather typical in character, developed, and ultimately death followed in six weeks. At the postmortem there was found an acute cystitis apparently secondary to a process in the intestines. Examination of the blood in life showed the bacterium coli, which was proved both by cultures and by animal experiments.

Examination of the central nervous system and of the peripheral nerves showed a polyneuritis parenchymatosa, and changes in the white and gray matter of the cord

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(1) Deut. med. Woch., Sept. 5, 1907.

immediately adjacent to the arterial twigs entering it. The anterior and posterior roots in the cervical and lumbar region were degenerated, and the cells of the anterior horn were particularly affected. These changes were evidently due to the poisons circulating in the blood, as proved by experiments upon animals, and the long course wholly without fever.

A. Porot and J. Froment[1] report two cases of multiple neuritis occurring concomitantly with cirrhosis of the liver, and call attention to the infrequency of such association, as compared with what our *à priori* conceptions would lead us to suppose likely. This form of association, besides being rare, is interesting in that it is practically restricted to females between the ages of 30 and 40 years, and in that it is found only with certain types of hepatic alteration, namely, the hypertrophic cirrhoses, to the exclusion of the other forms (atrophic cirrhoses of Laënnec). Moreover, certain secondary symptoms are also associated with it, thus giving to it a *clinical type,* not always rigorously outlined, but still sufficiently well so to make it easy to describe.

It is not necessary to detail the two cases reported. The first occurred in a young woman, 31 years of age, a wine-drinker, suffering with hypertrophic cirrhosis with ascites of rapid development, splenic enlargement, multiple neuritis, muscular atrophy, pain, and erythromelalgia of the lower extremities. The postmortem examination showed a large liver with diffuse cirrhosis and a little fatty degeneration, the typical lesions of multiple neuritis, and no lesions in the central nervous system. The second case occurred in a woman, 41 years of age, who had indulged in various alcoholic drinks to excess, but especially in wine. The illness began in May, 1906, with emaciation, loss of strength, repeated attacks of nose-bleed, and pains in the extremities. In November, 1906, disturbances on the part of the liver followed, the organ became enlarged and painful, and there were digestive disturbances. In December the legs became paralyzed, the arms paretic, and the emaciation extreme. There was transitory ptosis. Gradually the symptoms subsided, the emaciation ceased, the pains became less acute, and she began to walk once more. The liver

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(1) **Lyon Médical, No. 34, 1907.**

diminished in size, but remained at a line two or three finger-breadths below the costal border. It ceased to be painful. The case is reported in this state, with only a partial recovery of the symptoms.

The type above outlined briefly has been mentioned only once in the French literature, by Sainton and Castaigne. These authors basing their conclusions on four personal cases and four from medical literature, insist upon its rarity; but Porot and Froment believe such rarity to be more apparent than real, because most observers see and study only one of the constituent elements of this clinical grouping, but not both. Yet they willingly concede that cirrhosis and multiple neuritis rarely develop simultaneously with equal intensity.

As to *etiology,* those affected are always fairly young and almost always females. It is between the ages of 30 and 40 that acute alcoholism produces its maximum of organic disorders. As to sex, of the 18 recorded cases, 14 have occurred in women. As to the liver disorders, it is interesting to note that the atrophic cirrhosis of Laënnec is never the form found associated with the multiple neuritis. According to Sainton and Castaigne, the reason lies in the fact that those suffering from this form of cirrhosis have symptoms so alarming that they consult a physician before signs of multiple neuritis develop, and give up alcohol at once. The type of liver found is always the hypertrophic, in which the interstitial lesion and the parenchymatous degeneration go hand in hand. One may find, however, large fatty livers, pale, yellowish, of diminished consistency, in which fatty degeneration is intense, and the cirrhosis scarcely appreciable. Such types of liver are often found associated with marked delirium. And one may, on the other hand, find a type of liver in which there is marked interstitial sclerosis, with very little fatty degeneration, a veritable diffuse hypertrophic cirrhosis. No matter what the variety of hepatic cirrhosis, these cases all show splenic enlargement of a moderate degree.

The *sypmtoms* (a) on the part of the liver are, objectively, the enlargement of the liver, which may be excessive, the painful border of the liver on palpation, and rarely ascites, especially as a terminal symptom, and icterus. The hemorrhages deserve mention, especially the early nose-bleed, and the bleeding from piles, gums, pharynx, and esophagus. The symptoms (b) on the part of the nervous system are those of alcoholic multiple neuritis, the lower extremities being chiefly affected, both sensory and motor disturbances being prominent, the reflexes abolished, and vasomotor disturbances present. Pain is always a large element in the symptomatology, and usually sends the patient to the physician before hepatic symptoms develop. After the pain, paresis follows, then atrophy, at first in the lower extremities, and later all over the body. Of the vasomotor disturbances, local edema with redness and elevation of the temperature are noted. Sainton and Castaigne emphasize the involvement of the sphincters, but Porot and Froment see nothing unusual in their paralysis in cases with semicoma and mental confusion. Indeed, in these cases psychic disturbances are especially common. Sainton and Castaigne noted them in 5 of their 8 cases, and Porot and Froment find a still larger proportion. The psychosis found is Korsakow's syndrome.

The *prognosis* is usually grave, most patients dying in a few months. Death comes in coma with elevated temperature, a small pulse, vomiting, and the picture of grave hepatic insufficiency. The average duration is from four to eight months. The diagnosis is based upon the acuteness of the process, the intensity of the hepatic symptoms, and the signs of multiple neuritis. General paralysis of the insane will be differentiated by the hepatic symptoms, the absence of dysarthria, of pupillary disturbances, and the characteristic mental changes. The conclusions of the authors follow:

Cirrhosis and multiple neuritis very rarely develop simultaneously and with the same intensity in the same subject. When this association occurs, it does so with certain special characteristics. It develops almost always in women from 30 to 40 years of age. The atrophic cirrhosis of Laënnec is never found in association with multiple neuritis; instead, the cirrhosis is always of the hypertrophic and fatty type, large livers and fatty degeneration. The spleen is always, but moderately, enlarged. Hemorrhages, often early and persistent, are characteristic. Ascites is rare and slight, or else it is a terminal symptom. Icterus is less frequent. The multiple neuritis is of the alcoholic type, chiefly affects the lower extremities, is often very painful, and is accompanied by atrophy and vasomotor disturbances (erythromelalgia). It may extend to the upper extremities, and even involve the sphincters. There are always psychic disturbances, sometimes very intense. The prognosis is very grave. Death in three-fourths of the cases followed within from four to eight months.

Sensory and Trophic Neuritis. Felix Rose[1] presents a case of sensory and trophic neuritis following herpes, with trophic lesions affecting the bones of the hand, of a type resembling common rheumatism. The patient, a widow of 67 years, in Raymond's service, was seized on Sept. 17, 1907, with loss of consciousness. For a month previously she had had vague pains in the right arm and couldn't bear the contact of cold water with it. The unconsciousness came on sufficiently slowly for her to get back into bed (she had just risen), and it lasted about fifteen minutes. No paralysis followed, though a week of fever ensued, and the right arm pained. At the end of the week the outer surface of the arm from the thumb to shoulder broke out with a well-defined herpes, the pain being very severe at this time, rendering even the pressure of the clothing unendurable. At the end of a month the pains left, but were followed by severe pains in the hand, thumb, and index fingers. At the same time the edema shown in Plate VI appeared, followed five weeks later by edema of the forearm. At present her fingers are deformed and ankylosed, and are kept bandaged and warm, since warmth eases them. On Jan. 6, 1908, the edema extended from the back of the hand and the posterior surface of the forearm to a point above the elbow. The skin over the back of the hand is atrophic and somewhat pigmented, possibly from the application of drugs. The edema pits very readily. The articulations of the first and second phalanges of the fingers are hypertrophied, and there is an incomplete ankylosis of some of the joints. Thus flexion of the fingers, flexion and extension of the hand, extension of the forearm and abduction of the arm are imperfectly performed. Lumbar puncture was negative. A radiograph of the hands and wrists shows trans-

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(1) Nouvelle Icon. de la Salpêtrière, January-February, 1908.

parency of the digital and metacarpal epiphyses, and of the proximal epiphyses of the phalanges, due to physiologic senile rarefaction of the bones in both; but in the right hand there is in addition a very pronounced transparency in the phalanges of the thumb, and the first metacarpal bone. Moreover, the proximal phalangeal epiphyses are deformed and enlarged.

Despite the absence of electrical reactions of degeneration, the existence of a neuritis is unquestionable, and such neuritides are not rare after zona (herpes). The edema is a trophic edema, there being absolutely no signs of compression or blockade of the veins of forearm or arm.

RADICULITIS.

C. Mirallié,[1] in a general article upon radiculitis, first calls attention to its establishment on a sound footing by Erb and Madame Déjérine-Klumpke in the case of the brachial plexus, and the subsequent determination by Ferrier, Sherrington, and others of the motor and sensory supply of each root. The *symptomatology* is next discussed very thoroughly. Destruction of the motor fibers paralyzes and wastes the muscle-fiber which it supplies. The antagonistic muscles, not counterbalanced, next bring about vicious and permanent contractures, ultimately followed by actual shortening of the tendons. Electrical examination gives the characteristic reaction of degeneration. The distinctive feature of the process, however, lies in the fact that the group of muscles involved does not at all correspond to a particular peripheral nerve, but, instead, to a particular cord-level.

Antedating the motor defects are the sensory ones, and these are easily ascribed to their appropriate cord-levels. At the very beginning the patient complains of subjective disturbances in the territory supplied by the root involved, these being sensations of painful paresthesiæ, of weight, pins and needles, etc. At times these subjective sensations are interrupted by painful paroxysms, extremely violent, well illustrated by the lightning pains of tabes. Déjérine and his pupils have insisted upon an early premonitory symptom, consisting of a very severe pain, brought on

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(1) Le Progrès Méd., Jan. 11, 1908.

by sneezing. This "sneezing sign" betrays the hypersensitiveness of the roots during the stage of irritation. Later, when the stage of irritation gives place to destruction, the lightning pains cease, and alterations of sensibility appear. The peripheral nerves are painful on pressure, but not nearly so much so as in peripheral neuritis. This is because a given root sends its fibers into several peripheral nerves, so that a given nerve contains only a few diseased fibers.

Systematic examination into the sensory disturbances shows at first zones of hyperesthesia. These give place in time to hypesthesia and to anesthesia, and involve all kinds of sensibility: touch, pain, heat and cold, etc. Deep sensibility, muscular and articular, may also suffer, and even the stereognostic sense may be lost. Characteristic of all these sensory disturbances is their distribution, which is not that of a peripheral nerve, but is that of a particular cord-level, following the bands mapped out by Sherrington. Associated symptoms may be herpes, and exanthemata of radicular distribution. Lumbar puncture, if it discloses a lymphocytosis, indicates a meningitis as the cause of the radicular involvement.

As to *course,* some may be of abrupt onset, almost fully developed from the start, without premonitory phenomena; but as a rule they are slowly progressive. As to *distribution,* the radiculitis may be cranial or spinal; if it is the former, it as a rule involves the facial or trigeminal nerves; if the latter, it is apt to involve the roots going to the brachial or to the lumbar and sacral plexuses. It may, of course, involve the trunk or dorsal roots, and even the cauda equina. It may affect one or many roots, or predominate over the sensory roots or over the motor roots only.

The *diagnosis* is easy if the possibility of its occurrence is kept in mind. It is based exclusively upon the distribution of the sensory and motor troubles, these being located in the territory of supply of cord-levels rather than the territory of supply of individual nerves. Thus all cases of multiple neuritis are eliminated. It is really more difficult to distinguish between lesions of the cord and roots than between the roots and peripheral nerves. In this case the associated symptoms present in cord cases must be utilized, for the distribution alone affords no help. It is also wise to remember that a radiculitis may be the forerunner of grave cord disease.

Pathologically, radiculitis is merely inflammation of a root, and hence trauma of a root, such as rupture or tearing, is eliminated. Radiculitis is primary or secondary; primary when the root is involved during its passage through the pia mater; and secondary when a meningitis precedes it. Etiologically there figure all intoxications, especially syphilis, which is the cause in more than half of the cases. After syphilis comes tuberculosis, then meningitis and Pott's disease.

As to treatment, the etiology governs. If due to syphilis, an antisyphilitic treatment must be instituted, such as subcutaneous injections of mercurial preparations. If the lesion is very old a laminectomy may be tried. It often cures.

NEURALGIA.

At the French Congress of Alienists and Neurologists held at Dijon in August, 1908, the clinical forms and the diagnosis of neuralgia formed one of the two main topics for discussion. The introductory paper was read by Verger, of Bordeaux.[1] The old classification of Pitres was into (a) extrafascicular neuralgias, due to irritation of nerve-terminals; (b) fascicular neuralgias, due to irritation of nerve-trunks; (c) radiculomedullary neuralgias, due to irritation of the intraarachnoidean part of the nerve; (d) reflex neuralgias, in which the pain appears in the territory of a nerve other than the one actually involved; and (e) central neuralgias, which are really persistent hallucinations of pain, being to the sensory sphere what fixed ideas and obsessions are to the intellectual sphere. This classification, though serviceable, cannot be adopted in our practical nosology today, where we still deal with facial neuralgia, sciatica, intercostal neuralgia, etc. Yet it is valuable in that it replaces the conception of neuralgia as a disease entity by the conception of neuralgia as a syndrome, in which the clinical problem is an etiological and a

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(1) La Semaine Méd., Aug. 5, 1908.

topographical diagnosis. When such diagnoses are made, the treatment becomes easy.

For purposes of simplicity Verger therefore divides neuralgias into four groups: (a) those of facial origin, (b) those of the trunk, (c) those of the lower extremity, and (d) those of the upper extremity. Each of these has its acute, subacute, and chronic forms, and forms due to irritation of the ganglion or root of the nerve.

(I) Peripheral facial neuralgias. This includes all neuralgias due to some disturbance in the territory of supply of the trifacial nerves: teeth, mucous membrane of the mouth, nose, frontal or maxillary sinuses, the conjunctiva, or lachrymal ducts. Of these, the acute and subacute forms due to carious teeth or sinusitis are the more numerous. Sometimes these neuralgias follow grippe, malaria, tuberculosis, or syphilis. Most frequently they are supraorbital and transitory. Among the facial neuralgias are also those due to involvement of the Gasserian ganglion, and the so-called epileptiform type of neuralgia. The facial tabetic neuralgias are doubtless examples of the type due to involvement of the Gasserian ganglion, and the *tic douloureux* is the common name for the epileptiform variety, for the pathogenesis is unknown, and it is apparently independent of any peripheral cause. It is the most severe form of facial neuralgia met with.

(II) Thoracic neuralgias usually are due to direct irritation of the endings of the intercostal nerves, and are usually the symptomatic neuralgias of pulmonary tuberculosis or carcinoma of the breast. In many cases the local irritation seems to be in some abdominal organ. All of these neuralgias are reflex. Ordinarily their distribution is not exactly coincident with the territory of supply of the intercostal nerve involved. There are one or more zones, broader or narrower than the nerve supply, and occupying one or more intercostal spaces. Pressure over the spaces is painful, whereas sensation over the same territory is reduced to the prick of the pin. The pain is usually worse in the night than during the day. The usual reflex intercostal neuralgia is caused by affections of the stomach, intestines, uterus, or their adnexæ.

The radiculo-ganglionic thoracic neuralgias are met with in pachymeningitis, Pott's disease, lesions of the back-bone, or carcinoma with compression of the cord and roots. The neuralgia of herpes zoster is a pure example of the ganglionic type of intercostal neuralgia. These neuralgias due to pressure upon the posterior roots are very tenacious, but disappear when the roots are destroyed.

(III) Neuralgias of the lower extremities are found almost wholly in the territory of the sciatic. There are two groups: the sciaticas due to peripheral disturbances, and those due to disturbance of the roots and ganglia. There is still a third group, those due to no discoverable peripheral irritation, and which may be called "essential" sciaticas. The first group is due to pelvic or sacral tumors, gross lesions of the uterus and its adnexæ, neoplasms and inflammatory exudates along the course of the nerve, in the thigh or popliteal space. Sciatic neuralgias due to involvement of the nerve-trunk are apt to show (1) muscular atrophy, (2) trophic disturbances of the nails with vasomotor disturbances of the skin, (3) disturbances of sensation, and (4) diminution or abolition of the tendon-reflexes, especially the Achilles-jerk. This form is almost always due to an intoxication of some sort, such as alcoholism, joint-disease, gout, diabetes, or some infection such as grippe, tuberculosis, or gonorrhea. Diabetic sciatica is bilateral.

The radiculo-ganglionic type of sciatica shows zones of pain, according to the roots involved. Generally they are in the lumbosacral region, the gluteal region, or even in the perineotibial territory. The pain is both continuous and paroxysmal, nocturnal and diurnal, but the nocturnal pain is the worse of the two. Walking makes it worse, though it is rarely necessary to advise immobility. Sensory disturbances of the skin are common, but not constant, and are generally present in the form of reduction rather than absence of sensation. The localization corresponds with the roots of the nerves rather than with their trunks. In this form the knee-jerk is usually normal, while the Achilles-jerk is weak or abolished. Muscular atrophy is rarely severe, though frequently slight degrees of atrophy are met with. Lumbar puncture shows lymphocytosis of the cerebrospinal fluid in some cases. This form of neuralgia may usher in disease of the cauda equina, or carcinomata of the cord. Prognosis should be reserved.

(IV) Neuralgias of the upper extremities are rare, and are usually traumatic.[1] Some are, however, radicular. In the latter form the pain is acute, lancinating, sometimes intermittent, more frequently continuous, with violent exacerbations, day or night. Movement of the arm, forearm, or even of the fingers, and cough, all bring on paroxysms. There are no painful points of Valleix, and the zones of pain correspond with the roots rather than with the nerve-trunks. There is disturbance of sensibility, either hyperesthesia or hypesthesia, with radicular distribution. The tricipital and radial reflexes are normal. They are due to Pott's disease, or to spinal or cord disease, pachymeningitis, spinal tumors, etc. Sometimes they are due to specific spinal meningitis.

The author's conclusions follow: The conception of neuralgia as a morbid entity involving the nerves, is no longer in harmony with our present knowledge. We ought rather to consider that neuralgic pain constitutes a syndrome due to some involvement of the nerve anywhere from its meeting with the cord as posterior root to its termination in the periphery. There are two distinct clinical types of neuralgia, the neuralgia of peripheral origin, and the neuralgia of radiculo-medullary origin. They may be contrasted as follows:

NEURALGIA OF PERIPHERAL ORIGIN.	NEURALGIA OF RADICULO-MEDULLARY ORIGIN.
1. These differ according to the nerve territories involved.	1. As a rule these are identical in the different territories.
2. Pain is continuous, with exacerbations.	2. Spontaneous pain is particularly intense; it may be continuous, with paroxysms, or essentially paroxysmal with intervals completely free from pain.
3. The course of the nerves and the points of emergence of the peripheral branches are often pain-	3. Pain is not confined to the course of nerve-trunks, but is found in the territories which cor-

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(1) See Practical Medical Series, 1907, Vol. x, p. 180.

ful to pressure (Points of Valleix).	respond to cord-segments from which the roots spring.
4. Rubbing of or intense pressure upon the areas spontaneously painful, calls forth paroxysms of pain.	4. The classic signs of pain evoked by pressure on the nerve-trunks or the point of divergence of their branches, are but slightly present, and are frequently entirely absent. Peripheral irritation does not bring out a paroxysm of pain.
5. Cocain injected into the painful spot over the course of the nerve-trunks, or at the point where the reflex attack of pain is called forth, temporarily controls the pain.	5. Cocain injected into the painful spot, or over the course of the nerve-trunks, produces no sedative reaction whatever.
6. Disturbances of sensation are not constant; when they exist, they affect a territory coincident with the supply of the nerve affected.	6. Disturbances of sensation are very frequent, and affect a territory plainly corresponding to radicular distribution.

The diagnosis of these two great classes of neuralgia is of great practical value, since it permits proper and exact therapy and prognosis.

Treatment. Erich Schlesinger[1] proposes the use of cold injections for the treatment of neuralgias. He uses normal salt solution cooled to 32° F., and in sciatica he injects it in the neighborhood of the sciatic nerve. He would regard it as a misfortune if he should inject the cold solution into the sciatic nerve-trunk, since he might thus cause grave motor disturbances. He therefore calls his injections "perineural" instead of intraneural. He claims that these injections instantly remove very severe pain. He has

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(1) Deut. med. Woch., Feb. 6, 1908.

given to 42 cases of sciatica 51 such injections, which proves that very few needed a second injection. Some cases of acute sciatica were cured with one injection, and also many chronic ones. He is of the opinion that this injection of cold is better than the use of stovain, cocain, or eucain.

Fischler is not specially enthusiastic about the treatment of sciatic neuritis or neuralgias in mixed nerves by the injection of alcohol.[1] After reporting eleven cases cured by such injections, he reports a case of sciatic neuritis which he treated by the injection of alcohol, in which a paralysis of the external peroneal nerve took place, requiring a year to cure. To this case he adds three others in which a degenerative toxic neuritis was caused. On account of his own case and these others he does not now treat sciatic neuritis by this method.

FACIAL PARALYSIS.

Dopter[2] reports a case of facial paralysis occurring in the course of ambulatory erysipelas. The erysipelas began on the left side of the face. A few days after its appearance, masked symptoms of meningitis supervened, accompanied by paralysis of the left side of the face, by paralysis of the muscles supplied by the left hypoglossal nerve, and by mydriasis of the right eye. Lumbar puncture showed a clear cerebrospinal fluid, running out under pressure, and containing numerous lymphocytes, mingled with some polynuclear leucocytes, denoting meningeal irritation. This case seems to explain the pathogenesis of the paralysis found after some cases of erysipelas. Such nervous complications are rare, and set in during convalescence, when they occur at all. There has been reported after erysipelas (1) paralysis of the oculomotor nerves with amaurosis; (2) atrophy of the optic nerve, with ptosis; (3) paraplegia; (4) paralysis of the muscles of the neck, with paresis of the muscles of the buttocks, thighs and legs; and (5) facial paralysis. The explanation has always been the impregnation of the nerve tissue with toxins from the streptococci, which, acting through the circulation, pro-

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(1) Muench. med. Woch., Aug. 6, 1907.
(2) Le Progrès Méd., Nov. 30, 1907.

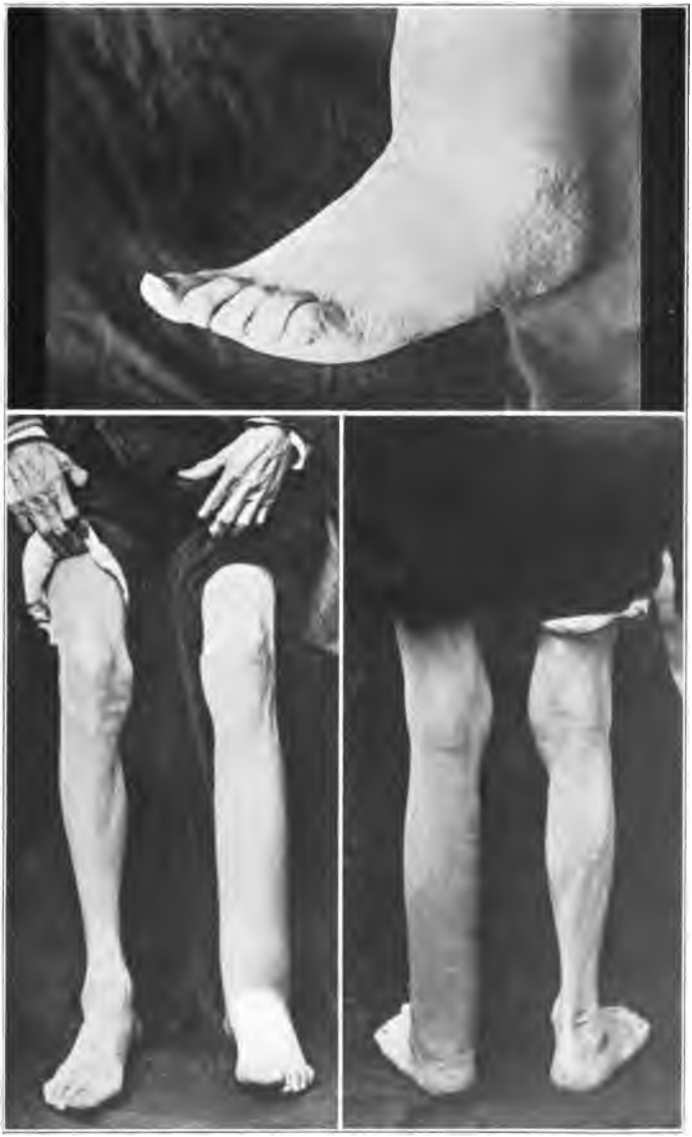

PLATE VII.

Chronic congenital trophedema.—V. Courtellemont (page 212).

duced distant disturbances, and which by acting directly produced paralysis at the site of the erysipelas. Dopter, however, believes this explanation to be wrong, and he advocates the hypothesis that these paralyses indicate a reaction on the part of the meninges. In this case there were the signs of a masked meningitis; headache, inequality of the pupils, Kernig's sign, and bradycardia. The examination, too, of the cerebrospinal fluid indicated irritation of the meninges. And the involvement of the 12th and 3d nerve in this case, together with the 7th nerve, supports this hypothesis. It is also supported by a case reported by Duroziez in which there was involvement of the third nerve, as shown by a double mydriasis and loss of the reaction to light and accommodation. There is not a true meningitis in these cases, but, instead, a meningitic irritation of a nature purely toxic.

A. Pitres[1] describes a case of nuclear facial paralysis of syphilitic origin. The patient, 49 years of age, denying categorically infection with syphilis, but confessing to heavy drinking, gave a history of a sudden attack of unconsciousness in 1896, not followed by paralysis of any sort, but followed by very violent headache in the right side of the head and face, vomiting, dizziness, and intermittent diplopia. Ten years later these symptoms, which had persisted with varying intensity, were definitely cured by twenty hypodermic injections of the green iodid of mercury. A year later the headache and vertigo reappeared, together with disturbances of vision, without retinal changes. Again he recovered. At the end of the year he awakened one morning to find the right side of his face pulled over to the left, with headache, worse towards evening and in the night. Moreover, there was vomiting. The facial paralysis was exactly limited to the muscles of the right side of the face, and these muscles showed the reaction of degeneration, having completely lost their response to faradism, and almost completely their excitability to galvanism. The pupils were found equal, with normal pupillary reactions, but with narrowing of both visual fields. There was also a marked paresis of the right external rectus muscle. There was no defect found in the

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(1) Jour. de Méd. de Bordeaux, Feb. 2, 1908.

fundus. Hearing was normal on the left and completely abolished on the right. The sensations of taste and smell were normal. There was some intellectual depression, a slight disturbance of speech, and a certain degree of dysarthria. His general condition was bad, his appetite poor, and he was habitually constipated.

On account of the associated deafness in the right ear, Pitres concludes the facial paralysis to have been caused by a nuclear lesion. Such a lesion would also cause paresis of the sixth and ninth cranial nerves, which in this case were involved. Headache and vomiting would doubtless be associated with such nuclear lesion so located. With specific treatment he made a perfect recovery.

Debove[1] reports a case of facial paralysis in a man in the secondary stages, showing the eruption. The paralysis readily yielded to specific treatment. He also records a case in a woman, who during the eruption stage showed facial paresis.

SYNDROME OF SCHMIDT.

Mouisset and Rouchut[2] report a case showing the syndrome of Schmidt, or involvement of the spinal accessory nerve. The spinal accessory nerve has an internal or medullary branch which innervates the soft palate, pharynx and vocal cords, and an external or spinal branch which supplies the sterno-cleido-mastoid and trapezius muscles. This internal branch joins the pneumogastric, with which it runs, and the combination by some anatomists is called the vagospinal, very properly. The *syndrome of Avellis* consists of a paralysis of the larynx and palate only, and results from a lesion of the nerve fibers which issue from the intermediate group of bulbar roots (between the plainly pneumogastric and the plainly spinal-accessory roots) and pass to the jugular ganglion. This amounts to a paralysis of the accessory portion only of the spinal accessory nerve.

The *syndrome of Schmidt* is the involvement of the whole of the spinal accessory nerve, both the accessory or medullary portion, and the spinal or cord portion. The case here reported is the sixth, five others having been gathered together by Le Meur in his thesis. The patient,

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(1) La Presse Méd., May 30, 1908.
(2) Lyon Med., July 19, 1908.

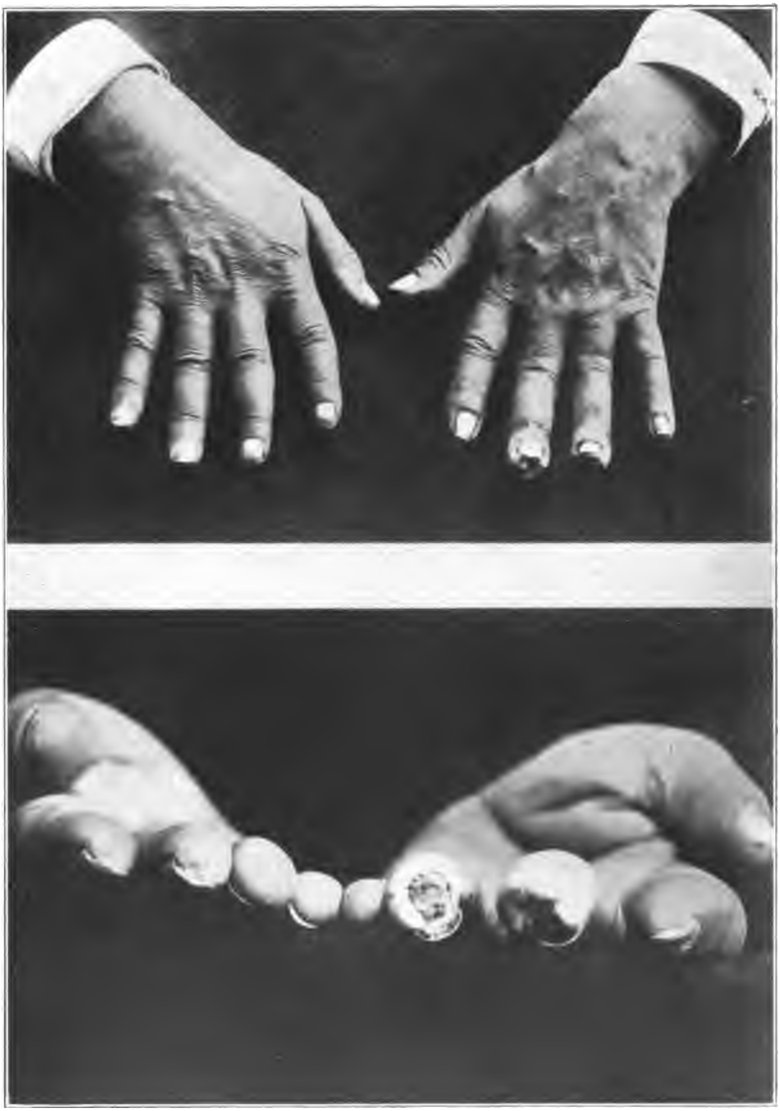

PLATE VIII.

Symmetrical gangrene of the extremities.—Raymond and Gaugerot (page 213).

a man 57 years of age, was a painter and plasterer. There was evidence of syphilis, but he had never been poisoned with either lead or alcohol. He was sent to the hospital as a case of narrowing of the esophagus, and he complained that for about two weeks he had not been able to swallow even liquids. The trouble had come on while he was in full health. The tongue was that of a syphilitic, fissured and sclerotic, but showing no atrophy or paralysis. The soft palate was asymmetric, being paretic on the right side. The right vocal cord was paralyzed. The radioscope showed a capsule of bismuth subnitrate to remain lodged at the upper part of the esophagus, though bougies could be readily passed. The right trapezius showed partial atrophy, and there was almost complete atrophy of the right sterno-cleido-mastoid muscle. Both muscles were paralyzed. In spite of such paralysis and atrophy, however, there was no vicious attitude, and he moved his head and neck very readily.

Syphilis being the evident cause, he was vigorously treated, with excellent results. The site of the disturbance cannot, in view of the symptoms, be cerebral, or bulbar, for the spinal cord supplies the sterno-cleido-mastoid and trapezius and presides over their nutrition. The trunk of the nerve must have been involved, probably by a basilar meningitis involving the vagospinal and the external branch to the two large muscles affected. The focus must have been intracranial, between the side of the bulb and the jugular ganglion, to give the syndrome described.

MISCELLANEOUS.

DYSKINESIA INTERMITTENS BRACHIORUM.

Oehler[1] reports an interesting case of dyskinesia intermittens brachiorum, which is to the upper extremity what intermittent claudication is to the lower extremity. The patient, 40 years of age, a mosaic worker, first noticed coldness in his arms mornings after starting to work. After an hour or two the feeling of cold disappeared, to reappear again the following morning. A few months later pain appeared together with the feeling of cold, chiefly in the

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(1) Deut. Archiv. für klin. Med., November, 1907.

flexors of the left forearm. A few days later violent pains followed in both upper arms. When the arms were forcibly extended the pains ceased. During the attacks the fingers were very pale and corpselike, and not a trace of pulsation could be found in the left brachial, radial or ulnar arteries. In the right arm the radial and brachial pulse could be felt, but the radial artery as such could not be made out. The electric reactions of the extremities were normal. The urine showed neither albumin nor sugar. There were no sensory disturbances in either touch, temperature or muscle and joint sense.

Only a few cases involving the arms have been reported. Nothnagel describes one due to an autochthonous thrombus in the right axillary artery in a girl 25 years of age. A few other cases are on record. In the present case the difficulty was not due to angiospasm, because, no matter what procedures were adopted calculated to dilate the arteries, such as the application of heat for a long time, by putting the arm in hot sand, or prolonged galvanization, the pulse never returned to the left radial or became any stronger in the right. The experiments indicate that arterial changes are responsible for this case, whereby the lumen of the artery was diminished. Yet the Roentgen ray showed no calcification of the arteries. Clearly in this case the disease has nothing to do with the nervous system.

TROPHEDEMA.

V. Courtellemont[1] reports a case of congenital chronic trophedema, several cases of which have already appeared in the Practical Medicine Series. (See Plate VII.) The author divides the cases of trophedema into four groups, as follows:

(1) A chronic trophedema, not congenital, but hereditary and familial. To this form belong the cases of Meige and of Lannois. The deformity involves several members of the same family, as a rule not appearing until puberty. It may be called *hereditary and familial noncongenital trophedema*, and its onset is at or just before puberty.

(2) A chronic trophedema, not congenital, not hereditary, and not familial. This is *acquired trophedema,* and

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(1) Nouvelle Icon. de la Salpêtrière, January-February, 1908.

appears at any time of life, after trauma, inflammations, or no cause at all.

(3) A chronic trophedema, both congenital, hereditary, and familial.

(4) A chronic congenital trophedema, neither hereditary nor familial. This is simple congenital trophedema, and it exists from birth in a single member of a family. This is the rarest type of all, and to it Courtellemont's case belongs. The patient, 83 years of age, has had the difficulty shown in Plate VII. all his life, being born with it. It never has interfered materially with the use of his leg.

RAYNAUD'S DISEASE AND ALLIED CONDITIONS.

Some cases of symmetrical gangrene, belonging to the type known as Raynaud's disease, are reported by E. J. Wood[1] as occurring in patients ill with malaria. [Numerous patients have been reported by French physicians from Indo-China and Algiers of Raynaud's disease and malaria, but this is the first series reported from America. From Wood's account, however, symmetrical gangrene is the better diagnosis, since the typical findings of Raynaud's disease do not seem to be present.—ED.]

Symmetrical gangrene of the extremities due to chronic obliterating arteritis of unknown origin is reported by F. Raymond and H. Gougerot[2] and is illustrated by the accompanying Plate VIII. In this case gangrene successively involved the right foot, the left foot, the right hand, and the left hand. It is preceded and accompanied by intense spontaneous pain, localized, continuous, and nonparoxysmal. It has been developing for seven years, by irregular exacerbations from one to six or seven months apart. It appears and disappears without any known cause, and each attack gets well spontaneously. There is first a thickening of the epidermis, which becomes blanched, then an ulcer forms which is very torpid. Ulceration is not preceded by local syncope or asphyxiation as in Raynaud's disease. The cause of the gangrene in this case is a chronic obliterating arteritis of unknown origin, involving not only the small vessels, but even the arterial trunks, the right radial having been permanently closed since 1905.

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(1) Jour. Am. Med. Assoc., Dec. 7, 1907.
(2) Nouvelle Icon. de la Salpêtrière, January-February, 1908.

No symptom of a neuritis is to be discovered. The case does not belong to Raynaud's disease, because there are no crises of pain, no syncopes or asphyxias of the extremities occurring paroxysmally. Moreover, the right radial artery is obliterated, and the left radial pulse has been gone a year.

PARALYSIS AGITANS.

Treatment. William N. Berkeley[1] makes a brief report of further experiences in the use of parathyroid gland for paralysis agitans. His process is as follows:

"Ox glands are gotten on ice fresh from the abattoir. They are carefully identified, trimmed with sterile instruments, pressed dry between folds of sterile gauze, minced in a small, finely cutting sausage machine (which must be scalded before using) and rubbed up in a sterile mortar with an excess of milk sugar. To this mass 1 per cent. boric acid is added and a trace of essential oil—usually peppermint. The preparation is dispensed in capsules, each capsule corresponding to ½ grain of fresh ox gland. The process calls for some technical skill in pharmaceutical work, but is the best I have so far tried. Of the hypodermic the dose is ½ c.c. per day. Of the capsules, 3 to 5 per day are enough. Both should be kept on ice. They do not appear to "go bad" in less than six weeks after making. The hypodermic injection should be given with rigid aseptic precautions. It works better under the skin than in the muscles. The site of injection is a little sore for half a day, but no abscesses have been reported, so far as I am informed. Veins should be avoided with special care, for most nucleoproteids tend to excite thrombosis when injected directly into the blood current.

"As regards the present status of work with a properly identified medicament, I should estimate that some seventy-five patients, in the hands of myself or of medical friends and helpers, have been treated. The remedy has been so far rather expensive and hard to get in large quantities. Many of the patients received it for a short while only. I now have notes or personal knowledge of thirty cases. Of these, two declined to continue the remedy; two have not been heard from; five denied any benefit; three showed temporary improvement only, and eighteen were progres-

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(1) New York Med. Jour., Nov. 23, 1907.

sively benefited during the entire period in which they were under treatment.

"If one should ask me whether, from the considerations just advanced, I believed I had proved a *specific nexus* between Parkinson's disease and the parathyroid glands, I should frankly confess the evidence quite inadequate. The gland material has a remarkable 'antispasmodic' action which may prove symptomatically helpful in numbers of different diseases. I do state, however, that no remedy so far suggested for paralysis agitans has had so happy or so permanent an effect. When a more perfect form of administration has been devised—possibly even a grafting of the fresh gland from one human being to another—the effects may be still more satisfactory."

MYATONIA CONGENITA.

A case of congenital muscular atony (myatonia, Oppenheim's disease) was reported in the Practical Medicine Series, Vol. X, 1907, p. 203, and a second case is now recorded by G. Leclerc as occurring in France.[1] Three other cases, one by Variot and two by Comby, were also presented at the Society of Pediatricians in Paris last winter. Variot's case was in an infant of 3 months of age, dying of broncho-pneumonia, showing postmortem remarkably pale muscles. At last report Comby's cases were improving with electrical treatment.

Leclerc's case is in a young girl, 4 years of age, born three weeks before full term, but showing no abnormalities. When she was 7 weeks old she had broncho-pneumonia and, presumably, attacks of laryngospasm. Following that the myatonia came to light. The child seemed paralyzed, incapable of executing movements, its head falling upon its shoulders, yet its facial muscles were intact. At present she walks as though she had a flaccid paraplegia. She cannot stand alone, or maintain the spinal column in an erect position. There is no paralysis, as can be determined when the patient lies down. There is no atrophy of the muscles, but they are very soft. The knee-jerks are absent, sensibility is normal, the sphincters are not involved. Though intelligence has not developed sufficiently rapidly,

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(1) Gaz. des Hôp., Dec. 10, 1907.

yet she is now learning to talk, and she understands what is said to her. She is, however, much less advanced mentally than she should be at 4 years. General hygiene, massage and electricity have been employed in this case. Possibly thyroid extract may be of some value.

FACIAL HEMIATROPHY.

T. Klingmann[1] reports a case of facial hemiatrophy which he correlates with 83 taken from medical literature. These cases he divides into two distinct groups as follows:

"1. Those cases in which the atrophy is complete without functional disturbance of the facial muscles, with little or no disturbance of cutaneous sensation and no vasomotor symptoms:—57 cases.

"2. Those cases presenting, in addition to the clinical picture of the first division, vasomotor symptoms, flushing or paleness, temperature differences and secretory disturbances:—26 cases."

The accompanying photographs (see Plate IX.) clearly illustrate the case here reported.

In this case the salient features are largely trophic in character; the discoloration of the skin, its diminished elasticity, the perceptible changes in the perspiration, and the atrophy of the subcutaneous tissue and bone, all point to a defective nutritive process. In the family history there was evidence of a neuropathic heredity and debilitating conditions. The author concludes that "from the statistical review it is evident that, in a majority of cases reported, the symptoms are those of disturbed trophic function; that the disorder is more or less acute and more rapidly progressive at the beginning of the disease, and that the association with acute conditions is a close one; traumatism is not infrequent, and infections are occasionally met with. In these points and in the close analogy of the symptoms with those occurring in neuritis there is evidence of a peripheral nerve lesion."

A case of *hemiatrophia facialis progressiva* is reported by J. R. Williamson[2] in a Hindu, aged 17 years, who ten months before had been the victim of an undiagnosed fever,

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(1) Jour. Am. Med. Assoc., Dec. 7, 1907.
(2) The Lancet, May 30, 1908.

possibly malaria or influenza. The onset of the hemiatrophy was four months after the fever. "Contrary to the majority of cases recorded, which, so far as I can ascertain, number about 100 in all, the skin was not hide-bound nor parchment-like in texture, nor was its color changed, nor did the eyebrow hairs fall out or lose their pigmentation; and sweating, which is usually diminished on the atrophied side, was in this case notably greater than on the unaffected side of the head. The bones had shared in the atrophy which had affected the skin and subcutaneous tissue and fat, but had apparently spared muscle tissue, as the patient could smile, show his teeth, frown, and masticate quite normally.

"The patient's ability to discern familiar scents appeared less acute on the left side, and he said that he did not recognize sugar when I placed it on the left half of his tongue. Whether these sensory impressions were really diminished is difficult to be quite sure about, as in persons of this class the intellectual appreciation of such differences is often very imperfect. As a rule such sensations have been unaffected. He complained of frequent neuralgic pains on the left side, especially above and below the orbit. In Romberg's case there was a history of very severe neuralgia which was subsequent to an attack of what was called 'facial erysipelas.' As far as I could ascertain there does not seem to have been any special skin lesion in this case. The patient did not suffer from any unilateral twitching. His pupils were normal and equal." The cause of the hemiatrophy in this case is as obscure as it is in all other cases.

MENTAL TORTICOLLIS.

Sicard and Descomps[1] report an excellent case of Brissaud's mental torticollis. The patient, 44 years of age, first experienced the torticollis two years before, just after the death of his wife. At first it was slight in amount, the head being turned a little to the left. Soon the condition became worse, and reached its present maximum about two months after its inception. He has learned, however, to correct the extreme position of the head; by touching

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(1) Nouvelle Icon. de la Salpêtrière, November-December, 1907.

with two fingers of his left hand the back of his head, he can make the spasm disappear. (See Plate X.) It reappears as soon as he takes his hand away. Thus the diagnosis of mental torticollis makes itself. It is simply an act of faith—of the faith which saves.

On entrance into Brissaud's service, a modification of the torticollis appeared. At times there were severe spasms which jerked back the head violently, putting it in forced extension, as seen in the plates. The external branch of the spinal accessory was exposed, and 90 per cent. alcohol was injected into it. Improvement followed and lasted only three weeks, when all the spasms returned, except that the torticollis gave place to "retrocollis." Further surgical interference being denied the patient, he went to another hospital, where the muscles of the neck were sectioned, the occipital portion of the trapezius, the splenius, the complexus, and the inferior oblique muscles being cut on each side. Figure D (Plate X.) shows the patient two months after the last operation, with the right shoulder greatly elevated, the head extremely retracted, and slightly turned to the left. The head, being deprived of its normal muscular supports, is with difficulty balanced on the spinal column. "In these cases," says Brissaud, "patience and the flight of time do more than forceful interventions or the craze for operating."

ADIPOSIS DOLOROSA.

Le Meignen[1] thoroughly discusses the disease known as adiposis dolorosa, or Dercum's disease, and insists upon its rather frequent occurrence. If all the cases reported are carefully analyzed, it will be found that only one symptom appears in every case, namely, adiposity. In the subcutaneous tissue there is a more or less thick, fatty layer, especially well developed at definite points. The places of election are the back, and particularly just beneath the junction of the neck with the trunk, the back of the arms, the upper part of the chest, and the anterior and inner sides of the thighs. Of course masses of fat occur elsewhere, but these are the usual sites. There is, however, a diffuse form, where the subcutaneous tissue is

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(1) Gazette médicale de Nantes, No. 23, 1907.

infiltrated generally, and sometimes to an extent so remarkable as to deform the limbs. Yet the hands and feet are usually exempt.

The skin has a peculiar appearance. It seems adherent to the tissue beneath, and resembles the skin of an orange. Palpation discloses beneath it small nodular masses, somewhat hard, and often painful. The pain, however, may not always be called forth by pressure, cases existing which are not associated with pain at all.

Besides adiposity and pain, two other symptoms may be noted as cardinal, despite the fact that they are not constant in every case. These are a more or less well-marked asthma and mental disturbances, ranging from slight neurasthenic symptoms even to insanity. Less usual but occasional symptoms are hemorrhages, such as nose-bleed, vomiting of blood, and subcutaneous ecchymoses; disturbances of sensation, especially subjective pains; arthralgias; ocular palsies; reduction of the intensity of smell, taste, and hearing; and finally certain trophic disturbances.

The syndrome develops very slowly, almost insensibly. The physician may be the first one to discover its presence, the patient merely believing himself "too fat." Le Meignen is certain that adiposis dolorosa is infinitely more frequent than is indicated by the number of cases reported. Systematic search for it made by him resulted in the finding of a relatively considerable number of cases. He finds that it predominates in the female, and at a mature age. Yet he has seen it among the young, even in girls of 15 or 16 years of age. Males are rarely affected, says Le Meignen, and but few cases in males have been reported. Contrary to the opinion of Eshner, he believes it to have no relation whatever to trauma.

As to *diagnosis,* two points deserve special attention: (1) the distinction between this form of obesity and the common form, which is sometimes hard to make, and (2) the distinction between it and myxedema. In ordinary obesity the fatty layer is more uniformly developed, and there are no pseudolipomatous masses, or points of special development of fat. In myxedema the skin is tougher, coarser, and is involved in a hard edema rather than in a specially well-developed layer of fat. Moreover, in myxedema the face is not respected, whereas in adiposis dolorosa it is. Finally, in young subjects, where myxedema may be suspected, the menses, instead of being delayed or suppressed, as in myxedema, are normal or increased in amount in Dercum's disease.

The *treatment* is still entirely empirical. Preparations of iodin, thyroid extracts, strychnin, electricity, Roentgen rays, hydrotherapy, have all been advocated, and all have met with occasional success in the hands of those who have used them. Yet according to Le Meignen, not one of these remedies has brought more than a temporary amelioration, much less a cure.

AMAUROTIC FAMILY IDIOCY.

Apert[1] contributes a very thorough article on amaurotic family idiocy, first described by Tay and Sachs. Curiously enough, the first case reported in France was as late as 1907, Apert and Dubois presenting the case. The author first gives a historical review of the disease supported by a fairly complete bibliography, and finds in the literature 82 indisputable cases, together with 27 cases in brothers, sisters, or relatives of the 82, not seen by the authors recording them, but appearing in the family history. Besides these cases there are a large number of aberrant forms.

Amaurotic family idiocy appears in the earliest years of life, but is not congenital, since the child afflicted with the disease is entirely normal at birth. In the great majority of cases the disease has begun in children between 3 and 6 months of age, onset in the second six months of life being unusual.

Symptomatology. The first symptom observed is apathy. The child gradually loses its vivacity, seems constantly half-asleep, hardly reacts to external stimuli, does not laugh, does not try to grasp things. He becomes incapable of sitting up in the cradle, or of holding up his head. He cannot hold things in his hand, but drops them at once. The movements made are slow and purposeless, and the reflexes are sluggish. Yet the electric reactions are normal and muscular force is conserved.

From the beginning vision is altered. The eyes fail to follow objects or to converge on a luminous point. The

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(1) La Semaine Méd., Jan. 15, 1908.

pupillary light-reflex is very sluggish. Examination of the retina shows a white patch in the macula, with a central spot of red. Later the apathy becomes intense, and the body is immobile and flaccid except for an occasional spasm of certain muscles. Sometimes there is a convulsion, the trunk stiffening, the extremities being in extension, the face reddened by suppressed breathing; in a minute or two the spasm is over and the child reverts to its customary appearance. Little is known of the sensibilities beyond the fact that mild pin-prickings are unheeded, more vigorous ones cause outcries, and severe ones cause generalized convulsions. The knee-jerks are usually exaggerated, as are also the Achilles-jerks, the elbow- and the wrists-jerks. Sometimes, on the contrary, the tendon-reflexes are abolished, as in Apert's case. The Babinski toe-sign cannot be obtained usually, but sometimes is positive. The sucking-reflex, swallowing, and respiration are not affected. Vision meanwhile grows more and more feeble until finally the spark of sight is wholly extinguished. At this period nystagmus begins.

In the final picture the child falls into a state of marasmus, the tendency towards continual spasm is marked, sometimes there is nystagmus, strabismus, spasmodic laughing, and disturbances of sucking and of swallowing. Emaciation goes on, the child living on his stored-up fat, the extremities and the trunk become extremely thin, the head, on the contrary, preserving its volume, so that it seems proportionately very much enlarged by comparison. Thus hydrocephalus has been simulated and confidently expected at the postmortem, where, on the contrary, it was not found. Death finally closes the scene, after fifteen to eighteen months of the disease. Thus the children seldom live to be 2 years of age, though those children in whom the disease begins late may live until the third year. About the oldest case reported (Koller) is 8 years of age.

Family Character. The disease is to a high degree a family one. Thus in 39 families having a total of 169 children there were 81 cases of family idiocy and 88 children free from it. Thus about half of the children born in families where the disease is found will develop the disease. Sex seems to be of no importance, except that perhaps the statistics show a slight preponderance of girls.

In four families consanguinity of the parents was shown. In many of the families there is a history of psychoses, neuroses, and mere nervousness in the ascendants and collaterals. Besides these nervous ailments, there is, in general, bad heredity as regards general physical condition. More remarkable than anything else in the heredity is the fact that all the cases hitherto reported have occurred in Jewish children. Among others the Polish Jews seem to have furnished most of the genuine cases.

The pathology is still under discussion, but is virtually settled. The important lesions are those of the nerve-centers, and are not to be found except by microscopic examination. The cerebral cortex, the central cerebral ganglia, and the anterior horns of the cord are profoundly altered. The lesions in general are less intense in the cerebellum and in the column of Clarke. The cell is swollen, doubled or trebled in volume, is globular in shape; the nucleus is crowded to the periphery, and the Nissl bodies are not regularly arranged concentrically about the nucleus. The neurofibrillæ cannot be followed into the interior of the protoplasm in the cells; indeed, they seem to be pulverized, their continuity thus being interrupted. The optic nerve is atrophic, its fibers are very slender, and but few are intact. The retinal cells are also markedly involved, especially in the *fovea centralis*.

Many aberrant forms have been reported as being cases of amaurotic family idiocy, the cases of Kuh and of Patrick, of Chicago, being placed by Apert in this category, because they do not show the white patch in the macula which he regards as a pathognomonic and essential symptom. Another group of aberrant forms is made up of the cases developing late. Of these aberrant cases, 9, among these the cases of Kuh and of Patrick, are placed very close to the typical cases. Patrick's case was of Christian parentage. Gordon's cases, reported in the Practical Medicine Series, Vol. X, 1907, p. 208, are regarded as departing widely from the typical cases of the disease.

As to treatment, Apert confesses there is none. Thyroid extract has been given without avail to his own patient.

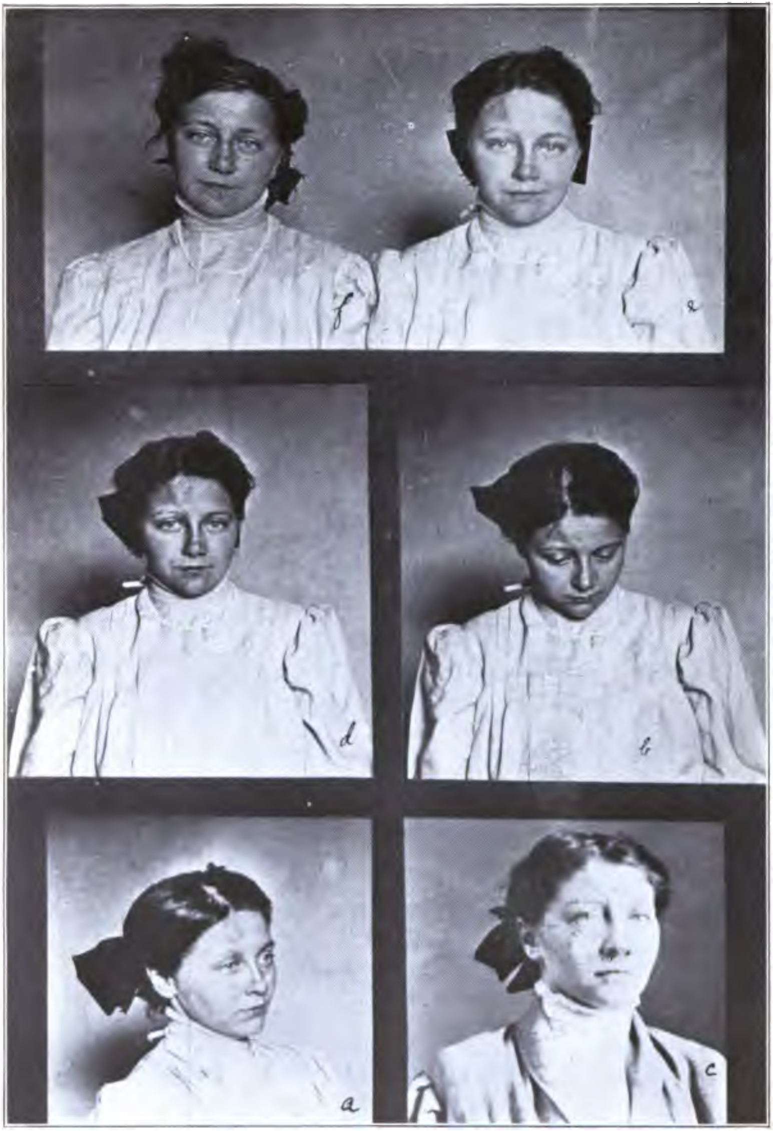

PLATE IX.

The photographs, a, b, c, d and e are of the patient and show the various conditions described in the text. The photograph is of the twin sister of the patient and shows slight asymmetry of the face.—T. Klingmann (page 216).

MENTAL DISEASES.

General Considerations.

Alleged Increase of Insanity. Noel Humphreys[1] discusses the question of the alleged increase of insanity, and his paper is ably summarized in the *Edinburgh Medical Journal,* October, 1907. "When it is baldly stated that by official computation the number of lunatics in England and Wales has increased in sixty-two years from 0.132 per cent. to 0.353 per cent. of the population, there seems, *prima facie,* some reason for supposing that insanity as a disease is becoming more prevalent; but on scrutinizing the figures more carefully we see that this conclusion is at least premature. For Mr. Humphreys points out that the figures in 1844 were admittedly imperfect. Based on a report of the Metropolitan Commissions of the Lord Chancellor, they did not include the large class of insane persons without certificate; even the returns concerning these private cases under certificate were very incomplete. Valuable as the return in question may have been for its purpose, Mr. Humphreys regards any comparison between it and recent figures issued by the Lunacy Commissioners as answering no useful purpose in proving or disproving the alleged increase of insanity.

"It is common knowledge that only a certain proportion of the mentally unsound come within the purview of the Commissioners of Lunacy, and this reserve of uncertified cases is a constant and variable source of error in attempts to estimate the prevalence of mental disease. The reports of the last four census enumerations, however, throw much light upon the varying number of this reserve. The first census in which mental defect was scheduled was that of

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(1) Jour. Roy. Statist. Soc., June 29, 1907.

1871, all forms being grouped together as lunacy, idiocy and imbecility, and the result was a return of 39,567 lunatics and 29,452 idiots or imbeciles. In 1881 and 1891, in answer to the same question, the total given rose first to 84,503, and then to 97,383; the proportions in the three census years being 3,039, 3,253, and 3,358 per million respectively. In the reports on the censuses, the opinion was expressed that the figures were probably understated, but that a nearer approximation to the true number was returned at each successive enumeration. In 1901 a change was made in the schedule, whereby the term 'idiot' was replaced by 'feeble-minded,' and in that year there was a very large increase (up to 132,564, or 4,078 per million) in the return of mental unsoundness. Mr. Humphreys draws the natural inference that this rise was due to the altered nomenclature having widened the sweep of the net; the return is probably more accurate, but has lost its value for comparison with previous censuses. The figures prove conclusively, however, that there has always existed, and that there still exists, a large reserve of mental unsoundness in excess of the constantly increasing number of the certified insane.

"From this reserve of uncertified mentally unsound persons the numbers of certified are constantly being recruited, and the very important question arises whether cases are now being certified which would formerly have escaped. Mr. Humphreys has compared the aggregate census numbers of insane persons with the numbers reported to the Lunacy Commissioners, and shows that the deficiency in the Lunacy Commissioners' numbers fell in 1871, 1881, and 1891, the percentages of deficiency being 17.8, 13.5, and 10.9; in 1901 the deficiency rose to 18.6 per cent.—presumably from the alteration in the form of question put. So far as they go, however, the figures suggest that year by year a larger proportion of all cases of insanity are certified under the Lunacy Acts. A further analysis of the census returns shows that beyond disclosing the existence of a large reserve of cases of mental unsoundness, they neither afford trustworthy evidence of the actual amount of insanity existing nor of its increasing or decreasing prevalence in the community.

"Much more important, however, than the declining rate

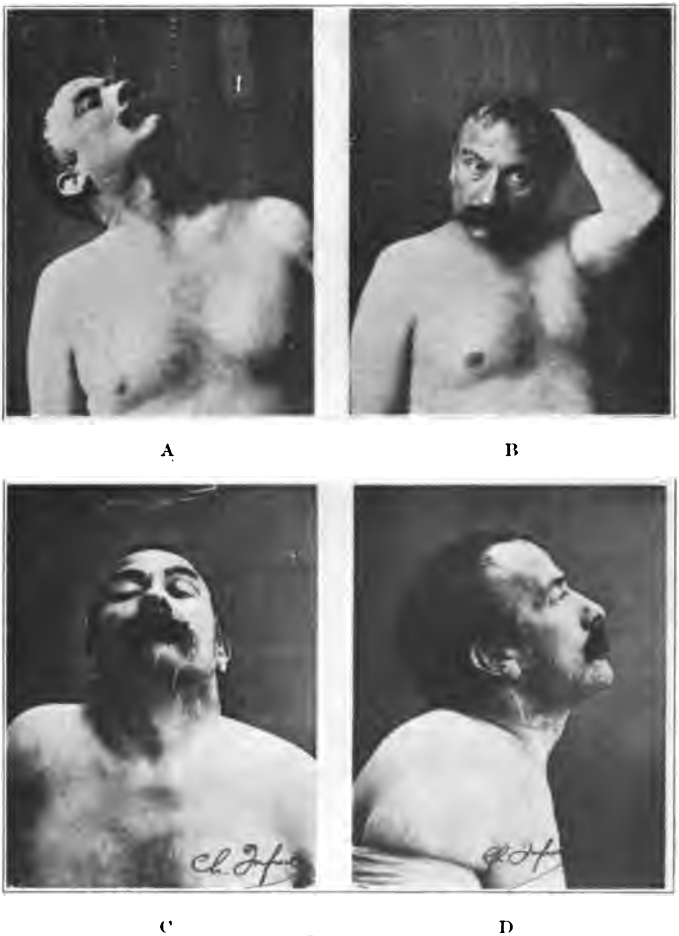

PLATE X.

Brissaud's mental torticollis. A and B illustrate the patient's attitude during the first period of the disease. C and D illustrate his attitude after the operation.—Sicard and Descomps (page 217).

of increase, and the absolute fall in the ratios of first admissions to the population, is what is known as the 'accumulation of the insane.' Those who assert that mental disease is really on the up-grade derive their sole support from the increased proportion, relatively to the population, of insane persons under the cognizance of the commissioners. Now, obviously the books of the commissioners may show more patients from year to year either (a) from an increase of new entries, or (b) from a decrease in the names removed. When fewer names are removed, the cases accumulate; names are removed either by death or recovery; hence a diminished death rate or a diminished recovery rate means accumulation—means a larger proportion of the insane.

"Besides accumulation, another very potent factor in increasing the insane population of asylums is the growing tendency to transfer cases of senile decay—harmless aged dements, and such—from workhouses to institutions. Taking admission to asylums from workhouses as a whole, a very large proportion (1,141 out of 1,455) was of persons above 70 years. The Asylums Committee of the London County Council have expressed the opinion that 'the certification of old people is a very important factor in the recorded increase of lunacy,' and that 'the great majority of old people who are received in asylums are in such a condition that it is possible to certify them as not of sound mind; but at the same time it is right to point out that in many instances this condition is the consequence only of natural decay through advance of years, and in many cases it is their physical rather than their mental condition which calls for most attention. This circumstance may explain why boards of guardians are so ready to send old persons who become troublesome in their infirmaries to asylums.' Another cause operating in the same way is the statutory grant of 4s. per week for pauper lunatics in asylums, which has much accelerated the transfer of harmless chronic imbeciles and lunatics not requiring the expensive treatment provided in asylums.

"The results of Mr. Humphreys' inquiry, therefore, discredit the prevalent assumption that insanity is on the increase among us. We have seen reason to suppose that the standard of mental unsoundness calling for certification has altered, and that cases are now registered which would not have been so certified half a century ago. Again, there is a large reserve of borderland cases, but, as the census returns compared with the Lunacy Commissioners' statistics show, this reserve is probably declining at the expense of a rising number of certified insane. People are now undoubtedly either more convinced of the advantage of asylum treatment, or less ready to incur the risks and discomfort of keeping mentally affected relatives at home—in any case, they send them in larger proportions to asylums. Here, then, is one great set of causes operating to produce an apparent increase of insanity. At the other end we have the accumulation of lunatics, as evidenced by the altering age distribution, the falling death rate, and the diminution of discharges on recovery. Finally, there is the tendency to certify, on financial grounds, simple cases of senile dementia for treatment in asylums. It is Mr. Humphreys' 'decided opinion that there is no absolute proof of actual increase of occurring insanity in England and Wales'; on the contrary, he believes that the 'increase of the registered and certified insane is not really the result of increased prevalence of insanity.'"

Etiology. (a) *Alcohol.* W. R. Dawson[1] discusses the effect which the abuse of alcohol has upon the mind. When alcohol is taken in small but continuous doses, mental degradation follows. Persons so addicted to its use become "coarser in the grain," lose the altruistic developments of the higher level, and almost invariably show a disingenuousness, a meanness, and sometimes an ill-nature which was once far from their character. "Egotism and bragging are common, and later, tendencies to grosser misdeeds, more especially of a sexual character, and suicidal impulses, may show themselves. In the intellectual sphere, failure of memory for recent events is a prominent symptom. Will-power is early and progressively affected, both in the direction of action and of inhibition, and it is this last which makes the reclamation of the chronic alcoholist so difficult. As time goes on, with deeper dissolution in

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(1) Dublin Jour. Med. Sciences, 1908, p. 412.

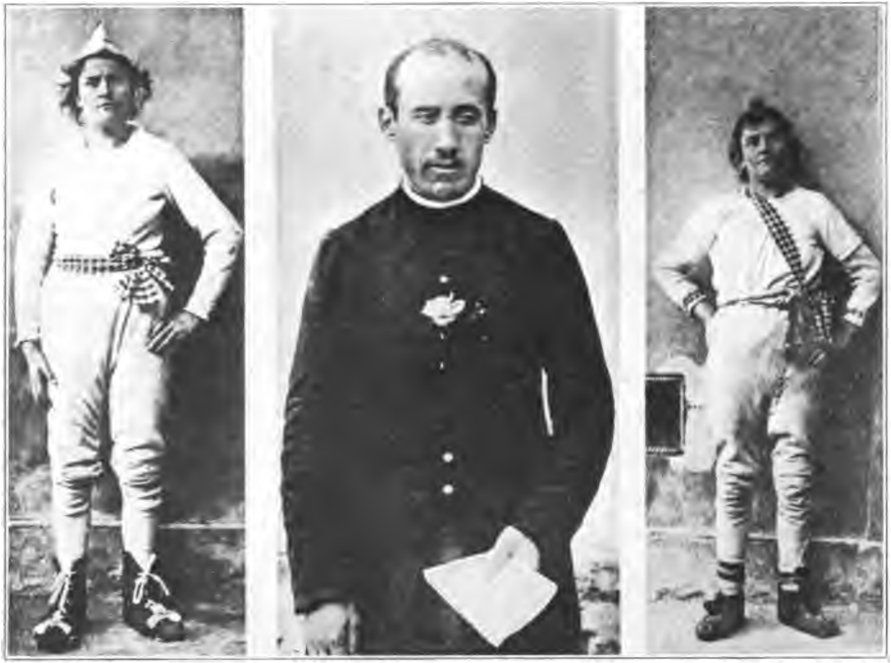

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PLATE XI.

Illustrating accessories of dress in manic-depressive states.—G. Boschi (page 236).

the intellectual and moral spheres, we have motor impairments, such as tremors beginning in the hands, loss of fine skilled movements as well as other defects of co-ordination, and reduction of actual muscular power; perverted sensations, such as flashes of light and cobwebs before the eyes, booming, whistling, buzzing, and like auditory phenomena, 'pins and needles' sensations in the skin, and so on; and, lastly, there are nutritional changes—faulty healing of wounds, susceptibility to the action of bacteria, obesity or emaciation, weak action of the heart, or even syncope. It is obvious that a person so affected is far from possessing a normal mind, and given a faulty diathesis and unhealthy surroundings, this mental abnormality is certain sooner or later to develop into technical insanity."

Statistics in Ireland show that in 1902, 10.4 per cent. of all cases were ascribed to alcohol, 7.9 per cent. in 1904, and 9.5 per cent. in 1906. Fallacies often occur, however, and thus the statistics are vitiated. Thus many insane people develop a craving for alcohol as an early symptom of their insanity. American statistics show that more cases of insanity are found among total abstainers than among moderate drinkers. Moreover, the distribution of the maximum of insanity does not coincide with the distribution of the maximum of chronic alcoholism. Indeed, in some places in England where drunkenness is most rife, there is the minimum of insanity. This fact has been brought out in maps showing the regional prevalence of insanity and drunkenness in England and Wales. In Ireland, Dawson believes that the proportion of cases of certified insanity in which alcoholism is the essential cause is considerably smaller than the official statistics would seem to show.

As to the forms of insanity produced by alcohol, it may be said that "some of them differ in no essential particular from insanity due to other causes, but in others certain symptoms are present which of themselves would suggest alcohol, notably a tendency to impulsive action, a suspicious habit of mind passing into delusion, and, especially in the more acute forms, the combination of mental confusion with hallucination which is more or less characteristic of toxic states in general. A very marked peculiarity of the chronic cases is the loss of memory for recent events, which, however, is shared with senile dementia. Alcoholics, however, have a most characteristic habit of supplying the place of memory by relating purely fictitious occurrences in good faith, and with a full belief in them, a peculiarity to which has been assigned the barbarous name 'confabulation.'"

The forms of insanity of most purely alcoholic origin are, as has been pointed out by Mott, delirium tremens, Korsakow's psychosis, and hallucinatory—confusional insanity, to which might be added some of the lesser degrees of alcoholic dementia.

In the neuropathic, whether congenital or acquired, alcohol produces its maximum effects upon the higher mental functions, the lower brain centers and the spinal cord being practically left alone. To this category belongs *delirium ebriosum* or *mania a potu,* a furious condition of wild maniacal excitement occurring chiefly in the young after moderate doses of liquor, and far outlasting the immediate effects of the drug. Less uncommon is chronic alcohol insanity, which may take the form of melancholia with morbid suspicions and fears, hallucinations, generally of hearing, and delusions, usually of suspicion and persecution, often connected with the digestive organs. These may be so prominent that the condition is sometimes described as alcoholic paranoia. In other cases there may be more excitement, and the delusions may take a more cheerful tone, such forms being designated alcoholic mania, and somewhat resembling general paralysis.

In alcoholic dementia, as indeed in the other more chronic forms of alcoholic psychosis which imperceptibly merge into the dementia, the mental symptoms are due to permanent degeneration of structure, not directly to alcohol, and somewhat resemble, as indeed do the pathological findings, what is met with in senile dementia. The marked symptom is, of course, loss of all recent memory, with the usual fabrication, general mental and bodily weakness, delusions, and hallucinations, the latter in part due to reduction and perversion of sensation. In some cases the delusions are grandiose, and the condition may bear a resemblance to general paralysis.

(b) *Influenza.* The psychoses of influenza are discussed by R. D. Brown.[1] He says that in the period from January, 1906, to the end of February, 1908, "twenty cases of insanity directly traceable to influenza were received into the Royal Edinburgh Asylum. Contrary to what is usually found, melancholia was not in excess of the other types of mental disease, there being ten cases of melancholia and ten of mania. Of the patients suffering from depression, seven were suicidal and one was markedly stuporose. Five cases exhibited very extreme agitation, restlessness, and restiveness, and labored under many distressing delusions of a persecutory character. Of the ten patients suffering from the manic form of insanity, nine were classified as acute mania, and in these there was complete or almost complete dissolution of mind, a constant incoherent chatter, with singing and shouting, while in seven there was great motor restless or violence.

"The maniacal type of mental disorder associated with influenza is more commonly seen in women and young people, and this comes on, as a rule, earlier than melancholia, which is present most frequently in men and elderly people. Of fifteen cases occurring in women in my series, nine were of the manic form of disease, while only six were melancholic, and of the five men affected, four were melancholic and one was acutely maniacal. In all the cases suffering from acute or delusional mania, with one exception, the symptoms set in within two weeks of the onset of influenza, four, indeed, beginning within five days of the commencement of the febrile attack. In the majority of the patients in the melancholic group evidence of mental disorder did not show itself in a pronounced manner until two or three months after the influenza, although in two cases it was said that the patients had never been quite well since the illness."

A marked feature of post-influenzal psychosis is the liability to relapse. This occurred in three melancholics and in six maniacal patients. The prognosis as to recovery is good, and twelve of the author's cases recovered. One died of acute mania and exhaustion, and one died of phthisis. Six still remain under treatment.

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(1) Scottish Med. and Surg. Jour., June, 1908.

SYMPTOMATOLOGY.

Hallucinations. J. H. MacDonald[1] has written a long article on the physiopsychology of hallucinations. After a brief historical review of the world's notables who were the subjects of hallucinations, he discusses the nature of the latter. He defines an hallucination as a subjective perception—that is, one without a corresponding objective stimulus. Hallucinations of hearing are the more common and important, particularly the hearing of spoken words. Occasionally the voices heard seem to come from the patient's own body, but more usually they come from without. Some hallucinations of hearing are pleasant, some very disagreeable, and some take the form of a command. Hallucinations of sight assume the most diverse forms, sometimes pleasant, sometimes grotesque, sometimes terrifying. Sometimes the forms presenting themselves are abnormally large (megalopsia), and sometimes proportionately small (micropsia). Hallucinations of smell, taste and touch are also common and diverse. In the sexual sphere there is a complex variety of tactile hallucinations. Often hallucinations of two or more senses coexist in the same patient. Sometimes hallucinations are unilateral in character, the voice always coming from one direction, or the vision always appearing to stand by the right side.

"When we come to inquire into the nature of the mechanism of hallucinations, their mode of origin, and site of production, we find ourselves confronted by a very difficult problem. On the whole, a careful consideration of all the facts warrants us in concluding that the theory of peripheral origin of hallucinations holds good in some, but by no means in all instances. In any case, we may agree that even when the primary stimulus occurs without the cerebrum, hallucinations are produced only after the sensory centers, which are the seat of the images, have been stimulated."

How do hallucinations influence the thought and conduct of the sufferer? Clearly they strengthen the insanity of which they are a sign. Sometimes they greatly stimulate the flow of ideas, but sometimes they execute vague fear and shut up the patient within himself. And the

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(1) Glasgow Med. Jour., December, 1907.

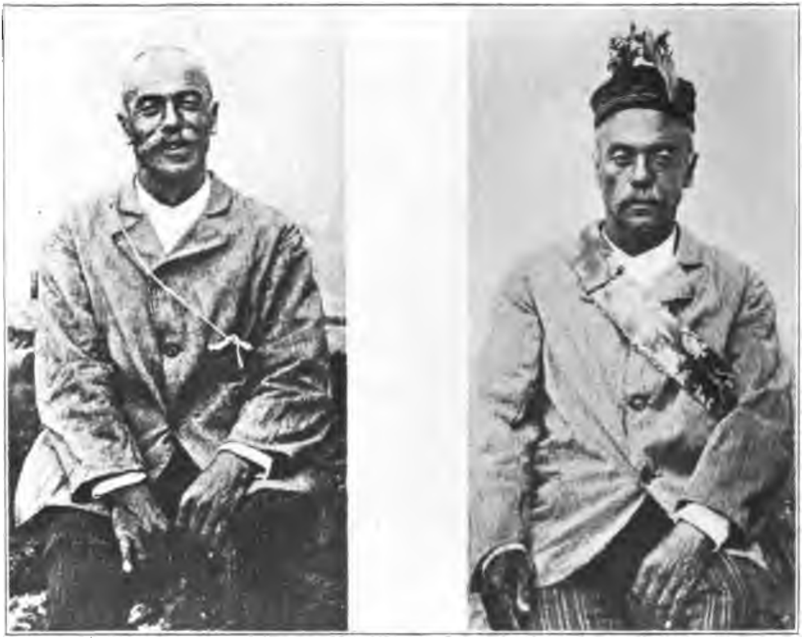

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PLATE XII.

Illustrating accessories of dress in manic-depressive states.—G. Boschi (page 236)

hallucinations naturally give rise to many vagaries of conduct. Are hallucinations compatible with sanity? "We know that they sometimes occur in individuals who recognize them to be false productions of their brains, and refuse to allow them to confuse their thoughts or to influence their conduct in an unusual manner. Ought such persons to be classed among the insane? I answer, No. It is true that hallucinations cannot possibly be regarded as physiological products, but departure from the type of physiological perfection does not in itself constitute insanity, else which of us could be counted sane? Few men are exempt from dreams, yet, strictly speaking, dreams represent a departure from the perfect physiological sleep. Who has not some mental peculiarity—some mental imperfection? And what of the hallucinations of those who have suffered amputation of a limb, yet feel as though it were still a part of their body? We do not call these men insane.

"After all, sanity and insanity are comparative terms. Each individual has a normal peculiar to himself, and applicable to none other. Any attempt to make a division between sanity and insanity must be artificial and conventional in the highest degree. The existence of hallucinations is certainly no criterion of insanity. We can, however, agree that when the subject of hallucinations fails to recognize these as anomalous productions of his brain, and allows his thoughts to be directed from their ordinary course, and his conduct to be regulated by the hallucinatory mental content, he is no longer of sound mind."

MANIA.

Etiology and Symptomatology. Lewis C. Bruce[1] devotes three extensive lectures to the cause and symptoms of mania. Mania, or states of mental excitement or exaltation, constitutes between 50 and 60 per cent. of all admissions to asylums in Great Britain. It may appear at any period of life, but in the vast majority of cases it is a disease of adolescence or early adult life. The disease conditions spoken of as mania may be divided, first, into mania with confusion—a state of delirious excitement in which the patient is wholly or partially unconscious of the

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(1) Edinburgh Med. Jour., February and March, 1908.

environment, and in every case of which confusion is a marked symptom; and, secondly, mania without confusion or the excitement of the *folie circulaire* type, more correctly designated manic-depressive insanity, from the fact that patients who suffer from this form of excitement are also liable to have attacks of depression. The excitement of manic-depressive insanity is characterized by elevation of mind, with little or no confusion. The patient keeps in touch with the environment, the special senses are hyperacute, and the chief mental characteristic of the disease is a rapid, ill-regulated, and easily disconnected train of thought.

As to etiology, Bruce believes that the diseases known as mania are conditions of brain poisoning, the poison or toxin in every case being a bacterial one. The bacteria causing these toxemias are probably streptococci, and the point of attack is almost certainly the intestinal tract. Possibly, "owing to some lowering of the bacterial defenses, certain strains of cocci become unduly increased in the intestinal tract. These cocci do not actually enter the blood stream, but they form toxins in the intestines which are absorbed by the blood-vessels and lymphatics in such quantities as to escape destruction in the liver and lymphatic glands, and they thus pass into the general circulation. These toxins have a selected affinity for the most highly developed nerve structures of the brain, to which they are carried by the blood-stream. When the toxin molecules are present in the blood stream in sufficient quantity to produce an acute brain intoxication, then an acute attack of mania is the outward result. When the poisoning is more gradual, then there is a gradual deterioration of the brain tissues, showing itself in eccentricities and changed character, which may lead finally to a chronic delusional state. The presence of toxin molecules, however, in the blood stream inevitably leads to the formation of antitoxin. The toxin molecules stimulate the cells of the body to throw out antitoxin molecules, which by combining with the toxin molecules render them inert. When a maniacal patient makes an apparent recovery, the antitoxin molecules have for the time neutralized the toxin molecules, and so we have a cessation of the symptoms. On the other hand, a lowering of the general bodily health or a failure on the part of the cells to form a sufficient number of antitoxin molecules, immediately allows the toxin molecules again to go free, further poisoning takes place, and another attack of mania is the result. The cause of the whole process, the streptococci in the intestinal tract, remains a source of danger, as they are unaffected by the formation of antitoxins, which cannot reach them in the intestine. This is not a mere hypothesis, because on examining the bacterial flora of the intestine in the cases of two patients who had recovered from confusional mania, I still found streptococci in almost as great numbers as when the patients were acutely maniacal."

MANIC-DEPRESSIVE INSANITY.

Kraepelin's manic-depressive insanity was one of the subjects set for discussion before the French Congress of Neurologists held at Geneva and Lausanne in August, 1907. Antheaume, who read the introductory paper, thus opening the subject for discussion to the Congress, first reviewed the matter of manias and depression states historically.[1] These states in French psychiatry were grouped under the periodic psychoses, the paroxysms or attacks appearing at intervals more or less apart, and very frequently many times during a patient's life. The attacks of mania or of melancholia usually reproduce themselves without regularity, after intervals of wide variation. There is also a wide variance in the duration of the several attacks. Besides, neither mania nor melancholia appears pure. It is the rule to observe, after a series of more or less long maniacal seizures, an attack of depression, or an access of mania may figure in the midst of a number of seizures of melancholia. Then there are alternating types of mania and depression, constituting the circular type of insanity. For all these, French psychiatry has adopted the term periodic insanity.

Kraepelin attacks this view, grouping all these forms under the head of manic-depressive insanity. Indeed, in this he also places simple idiopathic mania and melancholia, for he finds nothing in their symptomatology distinguishing them from the periodic types. Moreover, attentive observation of cases of mania and melancholia

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(1) La Semaine Méd., Aug. 7, 1907.

supposedly pure, will demonstrate signs of mania in depression, or signs of depression in mania. There is, too, no essential difference between mania and melancholia, as is shown by the mixed states, a large group of cases bridging the gulf between the two extremes. Kraepelin's manic-depressive type of insanity is characterized by three sorts of paroxysms, then: maniacal states, depressed states, and mixed states.

Although Antheaume does not formally adopt Kraepelin's view, he seems inclined towards it, for he confesses that it can hardly be maintained that there are such things as pure mania and pure melancholia. In the discussion Régis sharply defended the French view of mania and melancholia as morbid entity. Ballet objected to the term "manic-depressive" as being ungrammatical, but rather favored the idea for which it stood.

DEMENTIA PRECOX.

Symptomatology. S. E. Jelliffe[1] makes a few general reflections on the psychology of dementia precox. Symptomatically the most striking feature consists of the following:

"1. The occurrence of the reaction, for the most part, in the years about puberty, adolescence, and early manhood, i. e., within the years 18 to 28. This simply means that the majority of patients thus far studied fall within this period of life.

"2. The gradual development of a psychasthenic state which deepens into the sense of incapacity and inadequacy, and is but the beginning of a deteriorating process which shows itself in various attributes of the individual's mental powers.

"3. Thus a gradual emotional deterioration becomes apparent. This may be an indifference, or emotional stupidity, or may be characterized, as Stransky has so well shown, as an incongruity between the content of ideas and their natural emotional sequences.

"4. Modifications in the so-called intellectual sphere are apparent. While it is recognized that sharp distinctions between intellectual and emotional disturbances can

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(1) Jour. Am. Med. Assoc., Jan. 18, 1908.

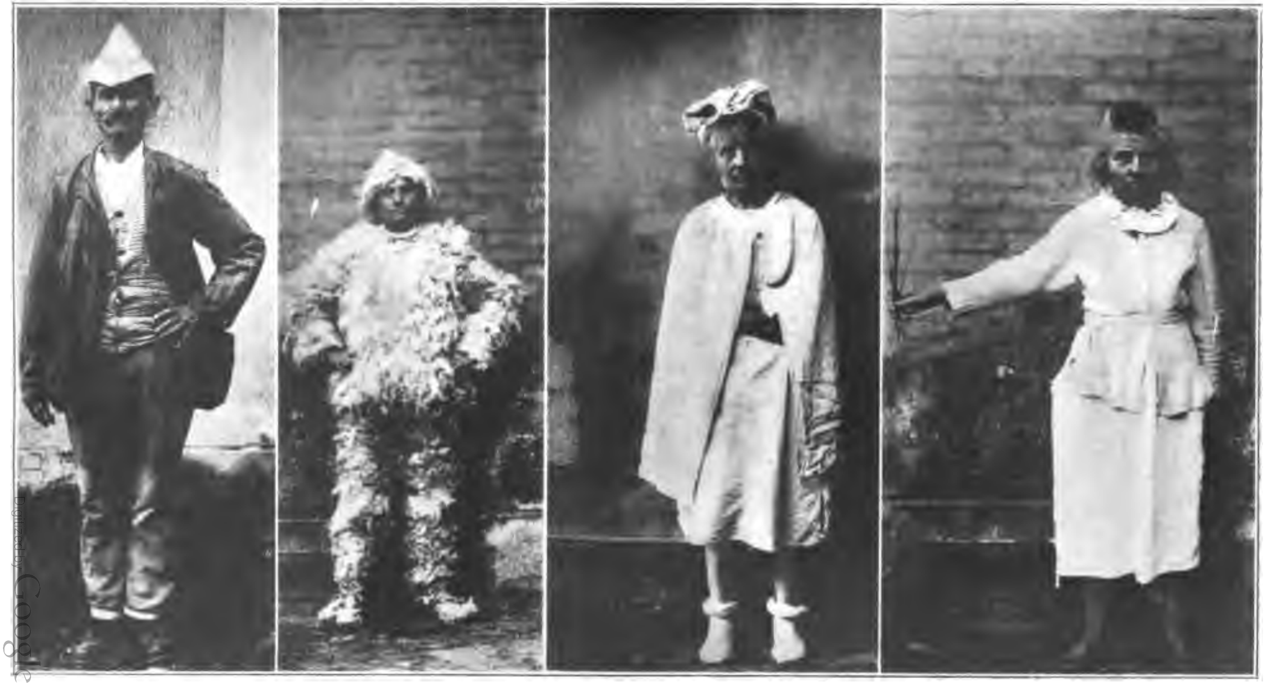

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PLATE XIII.

Illustrating accessories of dress in manic-depressive states.—G. Boschi (page 236).

be drawn only with the greatest of reservations, certain changes in the precocious dement are better grouped under this head than under others; thus, lucidity of consciousness, which is so often involved, at times leading to complete confusion; variation in the power of attention, a veritable attention disorder, both voluntary and involuntary; some modification of the faculty of orientation, often not marked; delusion development, often on an hallucinatory basis; the loss of apperceptive power and the development of the sense of unreality, of inadequacy, ideas of influence, of reference, and compulsory ideation.

"5. As a consequence of these and a number of other reactions there results a fairly constant and perhaps distinctive alteration in the character of the individual. Among the more constant and firmly established of the abnormalities of character are affectations, or eccentricities of manner; a tendency to search out striking combinations, neologisms in speech, bizarreness in writing, in artistic production, and unapproachability. The final development of the childish, foolish manner, so frequently seen as an end product, is best considered here perhaps as well. These affectations are particularly common in individuals of lower social strata.

"6. Finally, these patients show marked modifications in their motor conduct, i. e., in their general muscular reactions, both in the striped and unstriped muscles. Physical negativisms, stereotypies, affectations of posture and of bearing, catalepsies, automatisms, and a host of sympathetic phenomena are included.

"On a basis of variation in onset, in development, in the grouping, in intensity, and in persistence of these reactions, clinical psychiatry has erected certain types. Thus, those in whom the elements of early onset, slow advancing dementia, emotional deterioration, and attention disorder enter more slowly and persistently into the picture, we speak of as examples of simple dementia, heboids, or hebephrenia. With the introduction of more stormy intervals of great restlessness, much automatism, marked violence with stereotypies, verbigerations, negativism, etc., the group term catatonics is sanctioned; while in the paranoid group, which Kraepelin has so carefully erected, are arranged those who deteriorate very slowly;

whose character changes take place only after years; in whom the emotional indifference and attention disorder are very slight for years, but in whom the exaggerations and egocentric delusional interpretations eventually transform the character, and the exaggerations and eccentricities may take on a fantastic and almost artistic finish by reason of the very slow decay of the intellectual functions."

Accessories of Dress in Dementia Precox. Gaetano Boschi[1] calls attention to the curious accessories in clothing affected by patients with dementia precox and in the manic-depressive psychosis. These patients adorn their clothing with ribbons, flowers, and different decorative ornaments, intended to indicate some grade or profession, or convey some symbolic meaning. Melancholiacs and manic-depressive patients during the depressed phase of their malady never so adorn themselves. The first six figures in Plates XI.-XIII. are of manic-depressive cases, all showing symbolical ornaments. Thus the abbé in Fig. 1 wears a laurel-rose as a sign of charity; the patients in Figs. 2 and 6 wear paper hats, suggesting the soldier, and one of these, Fig. 3, also wears a band diagonally across his chest to indicate his rank as general. The patient shown in Figs. 4 and 5 wears adornments intended to show his rank as a general. A precocious dement, Fig. 10, wore for months a piece of string tied in a hole in his cap, saying that it served for respiration, and on another occasion declaring: "It is vision; it means the mind, the joy of man; we are formed of vision, that is"—when pressed for an explanation—"we manufacture air." A paranoid dement, Fig. 11, wore a little red cord on his breast which he explained to be "a sign which ought always to remain upon the side of the heart, to signify that man is red." On another occasion he said it meant that all men should be equal. The patient shown in Fig. 13 wore a small cloth sac, showing on the coat near the left pocket, and this sac he always kept full of paper. Occasionally he would gravely pull out a dirty piece and hand it to his attending physician as a telegram which it was important to send immediately. Figures 12 and 14 are merely military figures of the paranoid type of dementia precox. These military emblems are easy to understand,

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(1) Nouvelle Icon. de la Salpêtrière, January-February, 1908.

being very naturally present to the minds of the patients, and consequently readily adopted.

As between the dement and the manic-depressive type of patient, the former shows an absolute lack of rational meaning in his symbol, the explanation always being vague, or remotely applicable to the symbol. When this symptom is present, it is important in a differential diagnosis, but it is not constant. Another differential point is that a patient of the manic-depressive type will not wear the same emblem or strange costume for years, without change, while a dement will. Thus, Figs. 2, 3, 7, 8 and 9 represent manic-depressive cases, Figs. 2, 3, and 7 being of one patient, and Figs. 8 and 9 of another, the manner of dress suddenly changing at times. The manic case often forgets his ornaments, because he has other things to think of; the dement keeps fast hold of them, finding contentment in them. As to clothing, the dement tends to monotony and regularity; the manic-depressive patient, to multiplicity, disorder, and grotesqueness. Thus Fig. 7 has his clothing completely covered with little tufts of wool carefully sewed on; and no one but such a patient could invent the costume shown in Fig. 9, or carry so carefully the little bunch of straw. Oddities of costume serve to distinguish true mania from simple maniacal exaltation.

The author's conclusions follow:

(1) Accessories of clothing, observed very easily, may greatly facilitate the diagnosis; but in cases where the remaining symptomatology leaves the diagnosis doubtful they do not throw light upon the case.

(2) No form of ornamentation of the clothing is to be observed in melancholia or in the depressed state of the manic-depressive psychosis, or even in the maniacs of a sober mood.

(3) Distinction between accessories of costume with symbolical meaning and accessories of costume purely ornamental, is artificial.

(4) Either the former or the latter may be found in either psychosis, dementia precox or the manic-depressive type of insanity; in the manic-depressive type the badinage on the part of the patient in the attribution of symbolism to his own adornments is pathognomonic. Very often, the

interpretation given to the emblem by a dement is so absurd as to be pathognomonic.

(5) The clothing of the insane frequently takes a military turn.

(6) The changing of sex in the adornment of the clothing is more common in the female than in the male, and in the manic-depressive psychosis than in dementia precox.

(7) In the manic-depressive psychosis one may meet with a characteristic mutability in ornamentation and in dress; in dementia precox one may meet with a characteristic stereotyped preservation of a certain fashion of ornamentation or dress.

(8) Regularity and stereotypy in work of adornment is much more frequent in dementia precox than in the manic-depressive psychosis.

(9) Multiplicity and disorder, when found, is specifically characteristic of maniacal ornamentation.

(10) Abnormal adornments of costume are met with in the maniacal even when the disease has not yet reached a very grave stage; while in dementia precox such abnormal adornments attest a far advanced stage in the disease.

(11) There is a difference between the grotesqueness of the ornaments made by the maniacal and those made by the dements.

(12) Conditions of morbidity of a slight or marked type may be very favorable for esthetic manifestations, imperfect but vigorous. Study of the adornment of the clothing, a very complex matter, may throw some light on the problem.

(13) The ornaments of clothing of the insane are as a rule infantile or atavistic.

(14) Dull colors do not indicate any superiority of mind.

TREATMENT OF INSANITY.

Open-air treatment of insanity is advocated by C. C. Easterbrook, medical superintendent of the Ayr District Asylum, Scotland.[1]

"I. *The Asylum or Outdoor Exercise Treatment of Active Insanity.*—From 1894 to 1902 I followed the exercise system of asylum practice which has prevailed since

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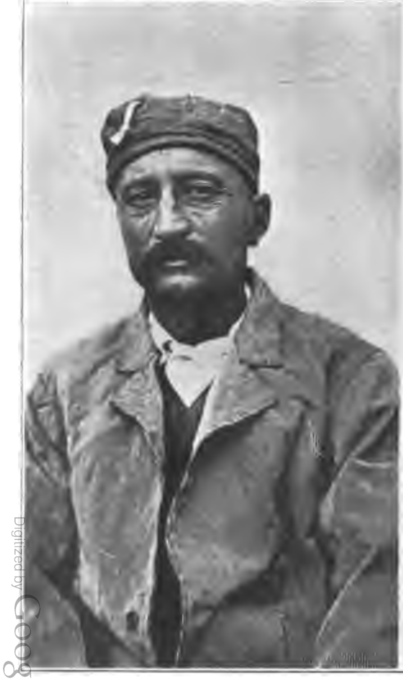
(1) Scottish Med. and Surg. Jour., November, 1907.



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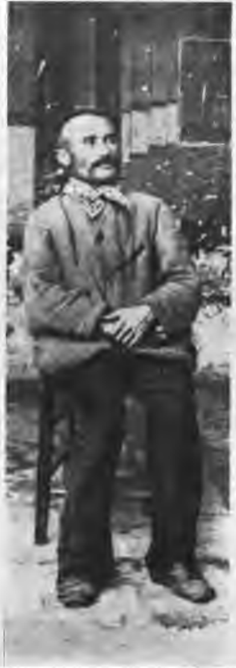

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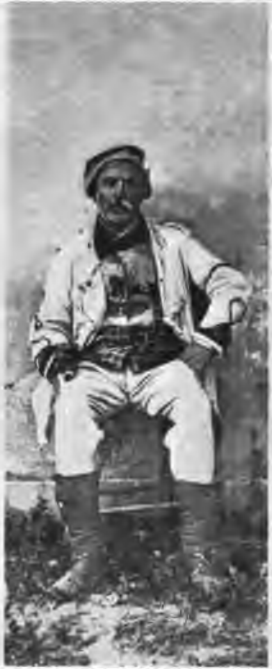

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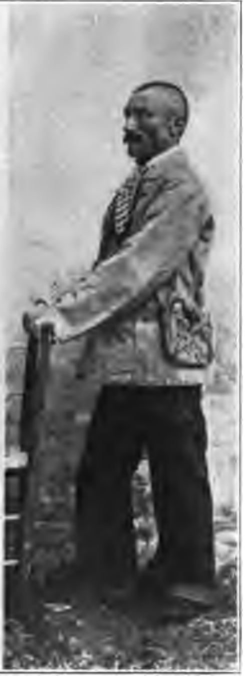

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PLATE XIV.

Illustrating accessories of dress in dementia precox.—G. Boschi (page 236).

the dawn of modern psychiatry, and still has many advocates, as a treatment of those actively insane. In my experience the intrinsic advantage of this system is the more or less rapid physical improvement of the patient, that is, as regards his skin, muscles, digestive and excretory organs, blood and circulation, nutrition and weight. This is followed by the improvement in the sleep and mental condition in the great majority of cases. That is to say, the improvement in the condition of the general bodily organs usually precedes the improvement in the state of the cerebral cortex and lower nervous centers. This retardation of the mental improvement, in my experience, constitutes the weak point of exercise in the treatment of active insanity, and is due, apart from the factor of the inherent powers of recuperation of the nerve centers themselves, not to the fact of the patient being out of doors, but to the effect of exercise in keeping up an excitation of the disordered nervous centers, and thus tending to consume unduly their diminishing chromatic substance and store of energy." The later researches of Mosso and others show that fatigue, whether produced by muscle or by brain work, is essentially a nervous phenomenon, an exhaustion and poisoning of the nerve centers. "Systematic outdoor exercise is therefore of doubtful utility in the treatment of active insanity, in which the finer symptoms of nervous initiation and exhaustion are so common. But just as rest and exercise are both necessary to the preservation of health, so are they the complement of one another in the treatment of disease, and carefully regulated outdoor exercise is specially useful during convalescence from active insanity."

"As to the recovery rate by the exercise treatment of active insanity, taking my own cases during the five years immediately preceding 1902, excluding transfers of all kinds, and including only those patients who had not previously during the existing attack been under treatment as certified insane persons, whether in institutions or under private care, including the good with the bad, both the highly recoverable cases, and the congenital idiots and imbeciles who came in with the others having become certified as insane for the first time in their lives, I obtained with 512 patients a recovery rate of 42 per cent., the results being slightly better with women than with men."

II. *The Hospital or Indoor Rest Treatment of Active Insanity.*—Since the summer of 1902 all new patients are received into wards of the hospital type, and observations are made daily and recorded on charts. This results in an enforced preliminary course of bed treatment for each patient. Moreover it gives the physician a better chance to learn his cases, and it lightens the work of the nurses. Moreover the patient resents being confined to a bed much less than he resents being confined to the asylum. The real value of the indoor rest treatment is the more or less rapid improvement in the mental and nervous condition of the patient, as shown by the lessened intensity of the mental symptoms, the motor restlessness and insomnia. There is also a gain in nutrition and weight. As to the recovery rate since 1902, Easterbrook gives 44 per cent., the results being slightly better with women than with men.

"III. *The Sanatorium or Outdoor Rest Treatment of Active Insanity.*—The sanatorium or outdoor rest treatment of active insanity is simply the hospital treatment by rest in bed carried out daily in the open air in verandas attached to the wards, instead of inside the wards and their bedroom as formerly. Since 1906 all newly admitted patients are placed straightway upon the verandas and treated with rest in the bed in the open air during daylight hours. The verandas are sheltered from the north, east, and west, and are 9 feet deep, thus permitting them to project well beyond the foot of the bed. The patients are in the verandas daily from 7 a. m. to 7:30 p. m. during the spring and summer months, when they receive all their meals out of doors. During the months of November, December, and January they are taken out to the veranda at half past eight o'clock, just after breakfast, and during the months of September to March they are brought in from the verandas in the evenings at 5 o'clock, just before the tea time (5:30 p. m.) of the day staff. Those patients who are sufficiently strong and exhibit no obvious or fine symptoms of fatigue, are allowed to walk to and from the verandas when the beds are being moved in the morning and evening, and to and from the ward lavatory as required during the day; but apart from this, such patients

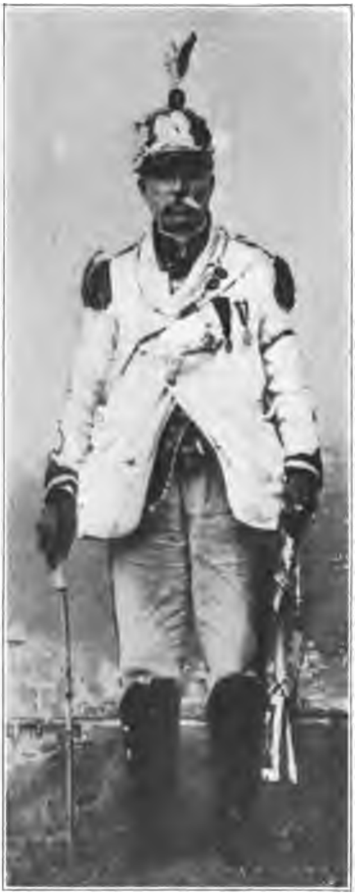

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PLATE XV.

Illustrating accessories of dress in dementia precox.—G. Boschi (page 236).

take no exercise during the period of rest in bed. Patients exhibiting signs of fatigue, weaker and easily exhausted patients, are wheeled or carried to and fro as required, and are allowed no exercise at all to begin with. Extreme physical weakness for obvious reasons contra-indicates the treatment. The severity of the mental symptoms is no contra-indication, and even in the worst cases does not prevent the treatment from being carried out straightway on admission with safety and propriety to all concerned, given plenty of nursing assistance, a sufficiency of bed and personal clothing for the patient and the means of isolation, which is secured at the hospital either by the use of screens between the beds in the larger verandas, or by using the smaller verandas such as are commonly occupied by phthisical patients undergoing open-air bed treatment." Even in cases of intense or fulminant excitement, the patient gets over the crisis more quickly than if other methods of procedure are followed.

The effects of rest in bed in the open air are first and foremost, a rapid subsidence of the active mental and nervous symptoms. Secondly, there is a rapid improvement in the physical condition. So excellent have been the results that relapses in resident patients are the signal for the out of door bed treatment, which is instituted *de novo*. Easterbrook, from his personal experience, finds that outdoor exercise benefits primarily the physical condition, but may retard the mental improvement; that indoor rest benefits primarily the mental condition, but may retard the physical improvement; and that outdoor rest benefits from the first both the mental and the physical condition.

The *duration* of the sanatorium treatment in the case of new patients naturally varies. In a small proportion of cases in which the bodily health is fair, and the mental and nervous condition is quiescent, the period need not exceed 2 to 3 days. Commonly a period of 1, 2, 3, or 4 weeks of sanatorium treatment suffices, and so in the great majority of cases the period of rest in bed in the open air does not exceed 1 month—a considerably shorter period than the 2, 3 and more months commonly employed by the chief advocates of the rest treatment carried on as hitherto indoors. The mental and bodily conditions each constitute the guide as to the duration of the treatment, and it may be stated generally that as soon as the active mental and nervous symptoms have subsided, and physical improvement has become established, as gauged especially by an increase in weight, the rest in bed stage ceases; but the open-air treatment still goes on, the patient being prescribed much sitting, with occasional short walks in the hospital garden, until convalescence is fully established, when exercise and work may be pursued with more freedom. During the after-treatment following the period of rest in bed the gain in weight continues to increase satisfactorily. The dietary enjoined during the sanatorium treatment is a simple, digestible, and moderately abundant one, comprising largely milk and milk puddings to begin with, and sooner or later light ordinary diet, with extra milk, eggs, and the like, the chief guides being the particular taste of the patient, and the state of the appetite, digestion, and weight.

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